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[*February* 24, 1956]

Hydatidiform Mole and Chorion-epithelioma—The Problem of the Borderline Case

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FOR many years obstetricians and pathologists working in the Orient have been impressed with the high incidence of hydatidiform mole and chorion-epithelioma amongst the Chinese. In Hong Kong, where the greater part of the Chinese population is derived from Canton and South China, the incidence of hydatidiform mole has been found to be 1 in 530 and that of chorion-epithelioma 1 in 3,708. These figures are derived from a comparison of all admissions of hydatidiform mole and chorion-epithelioma and all viable deliveries undertaken by the Obstetrical and Gynæcological Unit of the University of Hong Kong during a total period of twenty years. It should be noted that these figures do not include an additional 25% to cover the estimated incidence of abortions, as in Schumann's figures.

It is the object of this communication to summarize briefly the results of our experience in Hong Kong with hydatidiform mole and chorion-epithelioma, with special reference to the problem of the borderline case. Unfortunately all of our pre-war records were destroyed so that the material upon which this study is based relates only to the period January 1948 to June 1953, a five-and-a-half-year period, during which 64 moles and 15 chorion-epitheliomata were encountered. All of these cases have been carefully followed up and it therefore follows that the cases here reported have been under observation for periods ranging from a minimum of two and a half years to a maximum of eight years.

HYDATIDIFORM MOLE (64 CASES)

Age incidence.—The average age incidence was 29·7 years. Three-quarters of the cases occurred between the ages of 21 and 35, the youngest patient being only 18 and the oldest 49 years of age.

Parity.—The average patient had undergone 3 normal pregnancies, but only about one patient out of three gave a history of previous abortion. The abortion rate is usually in the region of 20% in Chinese patients, but in this particular group the previous history showed a rate of 11·7%. When, however, the molar pregnancy from which each patient suffered was included the abortion rate was immediately stepped up to about 31·5%. It is of interest to note that 4 patients had had no previous pregnancy whatsoever and 11 had had 5 or more normal pregnancies.

Amenorrhœa followed by bleeding was present in every case of the series. The average interval between the commencement of amenorrhœa and the delivery of the mole was 17 weeks, of which the last 5–6 weeks was complicated by continuous or intermittent bleeding. In some cases the bleeding was a minor or short-lived symptom lasting only a few hours, whilst in other cases it lasted as long as 17 weeks.

Toxæmic symptoms.—A special note was made of these, with the following results:

(1) *Hyperemesis.* There was a definitely increased incidence of vomiting, which was usually described as being much more severe and prolonged than in normal pregnancy. No fewer than 28 cases (or 44%) complained of this symptom.

(2) *Hypertension.* The average blood pressure in Chinese women is in the region of 110/70 mm.Hg, but there were 15 cases in this series (23·5%) where the blood pressure was above the 120/80 level, and in some cases it was as high as 180/110.

(3) *Albuminuria.* This was only present in 13 cases, or 20% of the series.

(4) *Œdema.* This was the least frequent of the toxæmic symptoms and was only observed in 8 patients, or 12·5% of the series.

(5) *Abdominal pain.* This was a fairly common symptom and was apparently associated with undue distension of the uterus. In some patients there was subsequent evidence of considerable hæmorrhage into the cavity of the uterus. The symptom was present in 23 of the cases, or 36%.

Size of the uterus.—In all cases except one the size of the uterus was recorded and classified in relation to the size usually considered normal for the duration of the pregnancy (Table I).

TABLE I.—SIZE OF UTERUS IN 64 CASES OF HYDATIDIFORM MOLE

Larger than normal ..	43
Normal in size	10
Smaller than normal ..	10
Unclassified	1
Total	64

TABLE II.—METHODS OF TREATMENT IN 64 CASES OF HYDATIDIFORM MOLE

Spontaneous evacuation followed by curettage ..	24
Dilatation of cervix and evacuation of the uterus	30
Vaginal hysterotomy	1
Abdominal hysterotomy	3
Hysterectomy without previous evacuation ..	6
Hysterectomy following previous evacuation ..	15

Biological tests.—Biological tests for chorionic gonadotrophic hormone in the urine were carried out before the evacuation of the mole in all but 11 of the cases, and in most instances frequent and repeated tests were performed to determine at what date subsequent to the evacuation of the mole the test became negative. The Friedman test was used in a certain number of cases, but the technique most frequently employed was the Galli-Mainini test, utilizing the local male frog (*Rana rugulosa*).

The frog test was found to be most useful for purposes of daily examinations both before and after the evacuation of the mole. It has proved to be both economical and reliable, particularly in cases where it has been necessary to follow the course of positive tests in high dilutions. In one of our cases as many as 99 tests were carried out during the process of investigation and follow-up. Each test involved not less than two frogs and when dilution tests were carried out at least six were employed. It is clear that the use of female mice or rabbits would be almost impracticable on such a large scale unless a very large animal house and considerable financial resources were available. The use of the male frog, however, during a period of over five years, has proved to be a cheap and rapid method, as well as uniformly satisfactory and reliable in its results. The test appears to be slightly less sensitive than the Aschheim-Zondek or the Friedman tests, and an occasional false negative has been encountered, but there has been no instance of a false positive reaction in any case of this series. The standard test employed was the injection of 5 c.c. of undiluted urine into the dorsal lymph sac of the frog. The appearance of spermatozoa in the urine within a period of one hour was regarded as a positive reaction. Dilutions of 1/5, 1/10, 1/50, 1/100, &c., were used as indicated.

In the 53 cases in which biological tests were carried out before the evacuation of the mole a positive result was obtained in every case. In 36 of these the test was positive in high dilution. A positive reading at a dilution of 1/100, 1/200 or 1/400 was frequently found. Several positive reactions at 1/800 were found, and in two cases there were positive findings at 1/2,000 and 1/5,000 dilution respectively.

Diagnosis.—Early diagnosis and treatment of hydatidiform mole is essential if the undesirable and dangerous complications which may follow it are to be avoided. In practice it was found that the main emphasis was placed on the following:

(1) Enlargement of the uterus out of proportion to the duration of the pregnancy (present in about 70% of the cases).

(2) Continuous or intermittent bloody discharge, starting at about the 12th week of pregnancy, usually not profuse and often brownish in colour rather than red.

(3) Absence of fœtal parts either on palpation or on radiological examination, even although the uterus may be enlarged up to the level of the umbilicus or higher. It might be mentioned here that in no case of this series was it possible to demonstrate fœtal parts by X-ray, and in only 3 cases of the 64 was evidence of the presence of a fœtus found when the mole was evacuated.

(4) The coexistence of toxæmic symptoms such as excessive vomiting, albuminuria, hypertension or œdema is very suggestive. Several cases showing signs of severe pre-eclampsia have been seen and one case of typical eclampsia was recently encountered, but this was subsequent to the present series of cases.

(5) The presence of a positive biological test for pregnancy in the patient's urine, particularly in a dilution of 1/50 or higher, with a rising titre is of the highest diagnostic significance.

Treatment.—Early treatment was instituted in nearly all of the cases in this series, the average time taken to reach a diagnosis being between three and four days. In a few doubtful cases observation for as long as ten days or even two weeks was necessary before a definite diagnosis could be reached, and even then (in occasional cases) it required a certain amount of courage to act upon the diagnosis. It may be said here, however, that in no case of this series was evacuation of the uterus unnecessarily or erroneously carried out. The actual methods of treatment employed have been summarized in Table II.

(1) *Spontaneous evacuation.*—Spontaneous evacuation, partial or complete, occurred in 24 cases. It is surprising that the diagnosis of a mole cannot be clinched more often by the spontaneous passage of a vesicle before the uterus is evacuated. Whilst spontaneous passage of the mole may often be hoped for, it can only be expected to take place in about one-third of the cases. Once the diagnosis has been made it is preferable to empty the uterus by more certain means and not to waste time in trying to stimulate spontaneous evacuation of the uterus by means of pituitary extract and other methods. In any case, spontaneous evacuation of the uterus should always be followed by curettage to ensure that the uterine cavity is completely empty.

(2) *Dilatation of the cervix and evacuation of the uterus.*—This was carried out in 31 cases, including one instance where vaginal hysterotomy was performed. It is undoubtedly the most certain method of treatment to dilate the cervix carefully to the point where it will admit a finger and then, after having separated the mass as far as possible with the finger, to evacuate the mole by means of a sponge-holding forceps. The use of the old-fashioned laminaria tent still serves a useful purpose in cases where the cervix is not easily dilatable with Hegar's dilators. The use of a slow intravenous drip of 10 units of Pitocin in 500 c.c. of normal saline is also most useful during the procedure of evacuation and will not only diminish the chance of serious blood loss, but will also minimize the risk of injury to the otherwise soft and atonic uterine wall. Preparations for the replacement of actual or estimated blood loss should always be in hand at the time of operation. Great caution should be used in curetting the uterine cavity for fear that the mole may be of the infiltrative type, in which case there may be little more than the thickness of the peritoneum separating the uterine from the peritoneal cavities. The gentle use of a blunt flushing curette with warm normal saline is permissible in most instances, however, and will usually ensure complete evacuation of the uterine cavity.

(3) *Abdominal hysterotomy.*—This is reserved for a few cases where the uterine enlargement may be very great or where the opportunity of proceeding immediately with hysterectomy may be desired, if this procedure seems to be indicated after the uterine cavity has been emptied and inspected. Hysterotomy was carried out on 3 cases.

(4) *Hysterectomy.*—This operation has been employed in three categories of cases. In the first category come those patients, who, by reason of their age and multiparity, are judged to be more suitable for hysterectomy as the primary form of treatment than for evacuation from below. The higher risk of malignancy in the elderly patient with a mole, particularly if she already has a large family, is the chief indication for the use of hysterectomy, and it applied to 6 of the cases in this group (average age 39·7 years and parity 7·7). In the second place come those cases where the presence of an infiltrating or invasive mole is suspected, usually on account of continued bleeding after evacuation or a persistently positive pregnancy test. There were 10 such cases in the present series. Finally there were those cases where definite chorion-epithelioma was found, and 5 of our cases were regarded as belonging to this category. These two latter groups form one of the main problems in the treatment of hydatidiform mole and they will form the subject of special discussion later in this paper.

Pathological examination.—Routine histo-pathological examinations were, of course, carried out in all of the moles which were evacuated. The amount of tissue removed varied in quantity from a few ounces of grape-like material mixed with blood clot to a large mass of tissue weighing 2 or 3 lb. The distinguishing macroscopic feature was the presence of innumerable clusters of vesicles varying in size from a few millimetres to as much as 30 or 40 mm. in diameter. In only 3 cases was an embryo or fœtus identifiable. In one of these cases the fœtus measured 35 mm. in length. In the other two the size was somewhat larger and in each case there was extensive hydatidiform degeneration of the placenta. No cases were included in this series where the diagnosis of mole depended solely on microscopical evidence. The microscopic features of all the cases designated as simple mole were characterized by the presence of vesicles of varying sizes, consisting of a poorly-staining stroma covered by chorionic epithelium with a varying degree of trophoblastic proliferation, especially in those portions of the mole in close relation to the uterine wall. Not infrequently molar tissue was found within the uterine blood sinuses, but there was no tendency in the simple case to penetrate through the blood vessels and infiltrate into the myometrium.

Subsequent course and prognosis.—The immediate results of treatment were good in all cases, in that there were no deaths as a result of the particular method of treatment employed. The subsequent course of each case was studied in relation to the following five factors:

(1) Disappearance or persistence of clinical manifestations such as bleeding, &c.
(2) Results of regularly repeated biological tests, first daily, then weekly, then monthly.
(3) Correlation of the two factors just mentioned with the pathological histology of each case, checked by repeated examination by curettage if indicated.

(4) Incidence of further pregnancies or of recurrent mole formation.
(5) Incidence of future complications, particularly of chorion-epithelioma.

Table III shows the rough direction of the subsequent course in the 64 cases of this series, based on follow-up examination s ranging from two and a half years to eight years.

TABLE III.—INCIDENCE OF COMPLICATIONS IN 64 CASES OF HYDATIDIFORM MOLE

	Number	Per cent
No complications ..	50	78·1
Invasive mole	5	7·8
Potential malignancy ..	3	4·7
Actual chorion-epitheli-oma	6	9·4

TABLE IV.—PERIODS OF TIME AFTER EVACUATION OF MOLE FOR TEST TO BECOME NEGATIVE

	Cases
Within the first five days	15
Within the sixth to tenth days	15
Within the eleventh to fifteenth days ..	10
Within the sixteenth to twentieth days ..	3
	43

There were 50 cases in whom the subsequent course was smooth and in these there was a rapid return to normal following treatment, without any future recurrence of unfavourable manifestations. Follow-up examinations demonstrated, in the great majority of cases, an early cessation of vaginal bleeding and a rapidly disappearing urinary content of chorionic gonadotrophin, followed by the resumption of normal menstruation. Perhaps the most important single criterion upon which the prognosis depends is the biological reaction of the urine and a special study was made of the length of time necessary for this reaction to become negative following the removal of an uncomplicated mole. In 43 of the cases daily biological tests were carried out on the male frog after the evacuation of the mole, and it was found that the ordinary standard test became negative at some point between the second and the seventeenth post-operative days. In some cases a concentration test would still produce a positive result for some days after the standard test had become negative, and in a few cases there would be an occasional recrudescence of a positive test at any time during the following two months. But for practical purposes the disappearance of a persistently positive pregnancy test with the standard amount of urine may be looked for within a period of less than one month after the evacuation of an uncomplicated mole. Persistence of a positive biological reaction after a period of one month is looked upon with the gravest suspicion. In the 43 cases which were specially studied it was found that the test became negative within 5–20 days after the evacuation of the mole (Table IV).

In 23 patients (out of the 42 uncomplicated cases who retained their uterus) no fewer than 29 normal pregnancies have occurred during the follow-up period and have been allowed to proceed normally. 4 patients have had two pregnancies each and one has had three. In none of these cases was there any history of serious abnormality during the pregnancy. There was no case in which the patient had a repetition of the molar pregnancy. In one, however, a patient developed chorion-epithelioma and died eleven months after a normal delivery which she had subsequent to a hydatidiform mole.

There were 13 cases, apart from the one just mentioned, in whom the progress following the evacuation of the mole was not satisfactory. In all of these there was either a failure of the chorionic gonadotrophin to disappear from the urine or a recurrence of a positive pregnancy test after a short free interval, and in most of them there was a persistence of irregular vaginal bleeding or the reappearance of such bleeding following a temporary cessation. Hysterectomy was carried out in all of these cases and revealed an infiltrating form of mole in 5 patients and chorion-epithelioma in 5. In the remaining 3 patients a condition very suggestive of early malignancy was found. These cases will be discussed later.

In this relatively small series of 64 cases of hydatidiform mole there was an incidence of 9·4% of chorion-epithelioma. This figure compares with Schumann and Voegelin's (1937) figure of 10% and Brews' (1939) figure of 8·3%.

CHORION-EPITHELIOMA

There were 15 cases in this group, of whom 12 died and 3 survive. There was a previous history of hydatidiform mole in 8 patients, of abortion in 2 patients and of normal pregnancy in 5 patients. The time interval which elapsed between the termination of the preceding mole, abortion or normal pregnancy and the establishment of a diagnosis of chorion-epithelioma varied from fifteen days to eighteen months, the average figure being just over five months. There was one exceptional case where the chorion-epithelioma did not develop until an interval of two years and nine months after the original mole.

Age incidence.—The average age of these 15 patients was 29·1 years, the youngest being 19 and the oldest 39 years of age. Contrary to the usual experience, no patient in this series was over 39, but this may be related to the fact that in patients over the age of 35 who suffered from hydatidiform mole it has been customary to carry out hysterectomy.

Clinical features.—

(1) *Vaginal bleeding*, irregular or continuous, and usually severe, was almost invariably the presenting symptom.

(2) *Hæmoptysis* was complained of by 5 of the patients, or 33% of the whole series.

(3) *A swelling* in the vulva was complained of by 2 patients, and an abdominal swelling by another.

(4) *The uterus* was enlarged in all of the cases, usually from two to four times the normal size.

(5) *Bilateral ovarian (luteal) cysts* were clinically obvious in 3 cases.

(6) *Metastatic growths* were clearly visible in the vagina or vulva in 5 cases, or 33%.

(7) *X-ray examination* of the chest showed multiple secondary deposits in the lungs in 9 of the cases, or 60%.

(8) *Biological tests for pregnancy* were done on 12 of the cases on admission and all were positive, some in dilutions up to 1/800.

Tests were not done in 3 cases who died shortly after admission as the diagnosis was not suspected in one case and animals were not available for the other 2.

Treatment.—It has been our practice in Hong Kong to carry out total hysterectomy, with bilateral salpingo-oophorectomy in all cases where chorion-epithelioma has been diagnosed, if the case is technically operable, even if obvious metastases are present in the lungs, vagina or elsewhere. There are sufficient reports in the literature of the subsequent disappearance of obvious metastases to make it worth while giving the patient the benefit of this admittedly remote chance. Brews (1939) has reported a fully documented case in which a vaginal metastasis disappeared completely after pan-hysterectomy and the patient remained alive and well over a year later, with a negative Aschheim-Zondek test. In the patients under discussion pan-hysterectomy was carried out in 7 cases. In the remaining 8 cases the disease was already too far advanced for treatment other than of a palliative nature. In one of these a laparotomy was done, but the involvement of the organs was already too extensive for removal of the uterus. In another patient radium was applied to a secondary mass in the vagina and in one other deep X-ray therapy was given to a pulmonary metastasis. The remaining cases already showed signs of advanced metastatic growth when they were admitted to hospital, and death was only a matter of days or weeks.

Results.—12 of these patients died and 3 survive. The relationship of the antecedent history to the prognosis is shown in Table V.

TABLE V.—15 CASES OF CHORION-EPITHELIOMA, PROGNOSIS IN RELATION TO ANTECEDENT HISTORY

	Dead	Alive
History of hydatidiform mole ..	5	3
History of abortion	2	—
History of normal pregnancy ..	5	—
Total	12	3

Each of the 3 survivors was treated by hysterectomy for chorion-epithelioma developing subsequently to the evacuation of a mole and the periods of survival have been seven and a half years, six years and three years respectively. They are all free from signs of recurrence and have had repeatedly negative pregnancy tests.

Fatal and rapidly progressive though this disease is in the great majority of patients, it is a mistake to believe that chorion-epithelioma is inevitably a fatal condition. In spite of Ewing's (1910) assertion that he was unable to find any record of operative cure of chorion-epithelioma, it would seem reasonable to suppose that there is a stage when the disease is curable by operation, and that if the patient can be brought under treatment at a sufficiently early date there is a reasonably good hope for a cure. Since this disease is most commonly preceded by hydatidiform mole, and since patients with a previous history of a mole are usually, if not invariably, followed up with greater care and suspicion than cases of abortion or parturition at term, there is little cause for surprise that the majority of the reported cures come from cases in which there has been a previous history of a mole rather than of an abortion or a normal pregnancy. Attention was drawn to this by Park and Lees (1950), who reviewed the outcome in 497 cases of chorion-epithelioma and showed that only 32% of the total deaths came from cases with a history of mole, whereas 68% of the deaths occurred in cases with a preceding history of abortion or of normal pregnancy. And this was the case in spite of the fact that approximately 50% of all cases diagnosed as chorion-epithelioma have their origin in a hydatidiform mole. Brews (1939), in his excellent study of 100 cases of hydatidiform mole and 24 cases of chorion-epithelioma, found that 6 of the 8 cases known to have developed chorion-epithelioma following hydatidiform mole survived for a period

of more than three years, whereas all 4 cases following normal labour proved fatal. The Hong Kong figures also illustrate the same facts.

The possibility of regression of pulmonary and vaginal metastases has already been touched upon, and there was one case in this series where a favourable result was hoped for. This patient, whose case is being fully reported elsewhere, was admitted to hospital in poor condition after repeated vaginal bleeding following the discharge of a hydatidiform mole two months previously. Examination of the urine showed a positive pregnancy test at a dilution of 1/200. Pan-hysterectomy was carried out and the diagnosis of chorion-epithelioma was confirmed. Multiple secondary deposits were found in the lungs on X-ray examination at this time. The disease was so extensive that deep X-ray therapy was considered inadvisable, and four weeks after the operation the patient was in a moribund condition with massive hæmorrhage into the right pleural cavity, causing deviation of the mediastinum to the left and intense dyspnœa. Both lungs were found by X-ray examination to be still further involved with metastatic deposits. After remaining in a critical condition for three weeks the patient began to improve and during the next seven months there was steady regression of the shadows in the lungs, until finally the shadows could no longer be detected. The pregnancy test also became negative, the patient gained weight and resumed her normal activities. Nine months after the hysterectomy, however, the patient had a sudden cerebral hæmorrhage and died within a few hours. Post-mortem examination revealed no gross evidence of chorion-epithelioma anywhere in the body. The lungs in particular showed healed scars consistent with complete regression of the metastases. Serial sections from the brain failed to show any secondary deposits of chorion-epithelioma, but it is difficult to believe that the fatal cerebral hæmorrhage was unrelated to the previous history of this patient. This case serves to demonstrate that there is a possibility that extensive secondary deposits may occasionally regress once the primary chorion-epitheliomatous growth has been removed.

In the 12 patients who died of chorion-epithelioma post-mortem examinations were obtained in all but one case. The primary growth was obviously in the uterus in all cases, with metastases (in order of frequency) in the lungs, brain, vagina, spleen, kidney, etc.

The prognosis, though admittedly poor, is not hopeless, and in this small series there has been a survival rate of 20%. This compares with Brews' (1939) figure of 33·3% and Novak and Seah's (1954) more recent figure of 17·5%. This latter figure is particularly significant since it embodies the results gleaned from the rich material of the Mathieu Memorial Chorion-epithelioma Registry and is based upon a study of 74 authentic cases of chorion-epithelioma.

The Borderline Case

The uncomplicated case of hydatidiform mole, and the typical case of chorion-epithelioma present no particularly difficult diagnostic problem, but in between these two clearly-established conditions there are many fine gradations where the innocent merges into the malignant by almost imperceptible degrees.

A large number of cases reported in the literature as chorion-epithelioma are, in fact, not genuine chorion-epithelioma but some borderline condition variously referred to as syncytial endometritis, chorio-adenoma destruens or invasive, perforating or malignant mole. Novak and Seah (1954), in their recent report, state that there were 74 undoubted cases of chorion-epithelioma in their material, but that they had excluded 85 cases wrongly diagnosed as chorion-epithelioma.

Hertig and Sheldon (1947) suggested the classification of hydatidiform moles into 6 different groups according to the benign, probably benign, possibly benign, possibly malignant, probably malignant and definitely malignant nature of the condition. The definitely malignant group (Group VI) was further subdivided into chorion-epithelioma in situ, syncytial endometritis, chorio-adenoma destruens and chorion carcinoma. There is perhaps a danger of obscuring by over-classification the very vital clinical problem as to whether or not a given patient has carcinoma, particularly as four of the conditions which can give rise to the greatest diagnostic difficulties are grouped together under the final heading of malignancy. The clinician is in the difficult position of having to make the final decision as to whether or not to take radical measures in any given case.

For purposes of the clinical and pathological study of the material here presented the following practical classification has been adopted:

(1) Uncomplicated mole.
(2) Borderline conditions:
  (*a*) Residual mole (including syncytial endometritis).
  (*b*) Invasive mole (chorio-adenoma destruens).
(3) Chorion-epithelioma.

Residual mole.—This term includes all residual trophoblastic proliferations within the uterine cavity other than those removed at the time of the original evacuation of the mole.

The term would therefore include portions of simple retained mole with their decidual attachments, examples of so-called syncytial endometritis or of locally malignant tissue. Diagnosis depends upon the pathological examination of any tissue removed by curettage subsequent to the original evacuation of the mole, and I have personally made it a practice to carry out a routine curettage one week after the removal of a mole in order to minimise the chance of further proliferation of any residual tissue which might still be capable of progressive change. Syncytial endometritis is rather an unsatisfactory term which tends to be used to describe conditions which cannot be regarded as simple residual mole and yet are not sufficiently clear-cut to be diagnosed as chorion-epithelioma. Novak and Seah (1954) define syncytial endometritis as "a residuum of trophoblastic cells after a normal pregnancy, abortion, or hydatidiform mole" and do not regard it as a neoplasm. At the same time it is well recognized that in certain cases which fit in with this description there is also some deep infiltration of trophoblastic cells into the muscle tissue of the uterine wall. It is, in fact, impossible to tell simply from the examination of the curettings whether or not the uterus is extensively involved by the mole or to exclude the chance that malignancy may be commencing to develop at a point within the wall of the uterus.

Case No. 168/48 (Path. No. 724).—Here the diagnosis rested between syncytial endometritis and chorion-epithelioma. In this case the pregnancy test became negative a few days following the evacuation of a mole, but reverted to positive twenty-two days later. Continuous vaginal bleeding also began again and there was a soft enlargement of the uterus. Pan-hysterectomy was carried out fifty-five days after the evacuation of the mole and a dark purple nodule was found in the posterior wall of the uterus, as seen in Fig. 1. Sections of this nodule showed areas of hæmorrhage and necrosis containing masses of active syncytial and trophoblastic cells, as seen in Fig. 2, and a diagnosis of chorion-epithelioma was made. The Friedman test became negative seventeen days after operation and the patient remains alive and well seven and a half years later.

FIG. 1.—Chorion-epithelioma (Path. No. 724) showing dark nodule about 2 cm. in diameter in posterior uterine wall. Well-marked bilateral lutein cysts are also present.

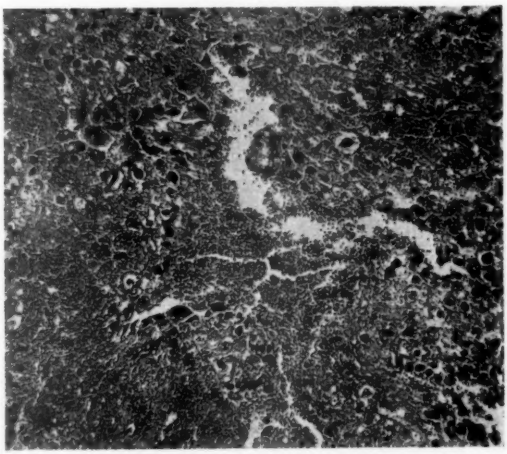

FIG. 2.—Photomicrograph of section taken from a portion of the nodule seen in Fig. 1, showing areas of hæmorrhage and necrosis containing masses of active syncytial and trophoblastic cells.

Invasive mole.—Perhaps the most important of the borderline conditions is the invasive mole, described by Ewing as chorio-adenoma destruens. Others have referred to this condition as malignant hydatidiform mole or as infiltrating, perforating, penetrating or destructive mole. In this condition the molar elements directly invade the uterine wall in one or more places and not infrequently a mis-diagnosis of chorion-epithelioma is made when a uterus is removed for this condition. The condition is by no means always malignant, however, and three varieties may be distinguished:

(1) *Simple invasive mole.*—The histological appearance of an invasive mole may show no difference whatever from that of a simple mole, except that it has invaded the myometrium. There may be slight or moderate hyperplasia of the trophoblast, but nothing more than is frequently seen in a simple mole and nothing which histologically resembles malignancy. Such a mole will, of course, persist after the intra-uterine portion of the mole has been

entirely expelled, and it may retain its activity without necessarily becoming malignant. Clinically, the patient may show no other sign than a persistently positive pregnancy test, which may be present in comparatively high dilution. There will probably be some enlargement of the uterus. Uterine bleeding is not always present since the mole may be entirely shut off from the cavity of the uterus. Furthermore, curettage, whilst it might yield a small amount of residual mole from the uterine cavity, would almost certainly fail to reach the infiltrating portions of the mole unless undue force were used, risking perforation of the uterus. It is in cases such as this, particularly in the instance of a young married woman with no living child, that the clinician is faced with a serious dilemma. If he performs an early hysterectomy he may find that the uterus shows no serious pathological changes. If he delays operative treatment, in the hope that the condition may regress and ultimately resolve, he may find, to his chagrin, when he ultimately decides to operate, that malignancy has already become established.

Case No. 424/53 (Path. No. 14902).—This patient, aged 31, had had 5 full-term pregnancies followed by a hydatidiform mole. The mole was evacuated and following this the bleeding stopped, but the pregnancy test remained positive. Two months later irregular bleeding began to occur. When the patient reported again, three and a half months after the evacuation of the mole, the uterus was found to be definitely enlarged with slight bleeding from the cavity and the pregnancy test was positive at 1/5 dilution. Curettage at this stage revealed nothing abnormal. But two weeks later the pregnancy test was positive at 1/20 dilution, and at this juncture it was decided to do hysterectomy. A large nest of active molar tissue, 2 to 3 cm. in diameter, was found in the uterine wall, beyond the reach of the curette, and completely covered over by muscular and endometrial tissue (Fig. 3). Sections showed a typical molar structure, with no suggestion of malignancy. The patient remains well two and a half years later.

FIG. 3.—Specimen of benign invasive mole (Path. No. 14902) removed at operation, showing nest of molar tissue buried in the muscle of the uterine wall and covered by an unbroken layer of muscle and endometrium. Curettage would obviously fail to reach this tissue.

FIG. 4.—Specimen of invasive mole (Path. No. 10527) as removed at operation, showing extensive invasion and destruction of the uterine muscle over the whole area of the fundus. The peritoneal layer in this situation was reduced to the thickness of tissue paper.

(2) *Invasive mole with signs of regression.*—Cases in almost every respect similar to the one just quoted may occur in which, when the uterus is removed, the mole is found to have penetrated the uterine wall, but is obviously in a regressive stage. In such a case a gradually weakening pregnancy reaction and a slight uterine enlargement may be the only positive signs. Undoubtedly there must be other cases, classified as uncomplicated moles, in which the mole has invaded the uterine wall, but has regressed to such a degree that the pregnancy test becomes negative at a relatively early stage following the evacuation. There is no doubt that many of the cases with an occasional or even persistent positive pregnancy test during the first month or more following the removal of a mole are really examples of this condition, but the diagnosis could only be confirmed by hysterectomy.

(3) *Invasive mole with signs of progression.*—In dealing with this condition there are usually obvious indications that all is not well with the patient following the evacuation of the mole. In these cases there is deeper penetration of the uterine wall and the molar process may perforate through the uterine wall into the broad ligament or even into the peritoneal cavity. There is usually appreciable uterine enlargement in these cases with

uterine bleeding and a definitely positive pregnancy test, often in high dilution. Early hysterectomy is indicated in such cases since there is a risk of internal hæmorrhage quite apart from the risk of malignancy.

Case No. 346/52 (Path. No. 10527).—This patient, aged 24, had had one normal pregnancy followed by a mole, which was passed spontaneously. Curettage following this showed normal and degenerated villi, and in some places a very active trophoblastic layer, but no evidence of malignant change. Uterine hæmorrhage and a repeatedly positive pregnancy test were present for the next two months, and the uterus remained soft and bulky. Since the pregnancy test was positive in increasing dilution it was decided to do hysterectomy at this stage, and it was found that the fundus of the uterus was invaded by active molar structures almost to the point of perforation (Fig. 4). Sections showed extensive invasion of the uterine muscle and cellular changes strongly suggestive of chorion-epithelioma, but on account of the presence of numerous villous structures and the relative scarcity of mitotic figures it was decided not to call this condition malignant. The pregnancy test remained positive for nearly two months following the operation, after which it became negative, and the patient is alive and well three and a half years later.

Case No. 405/53 (Path. No. 14707).—This patient (a 13-para, aged 41), had a very active invasive mole which perforated through into the broad ligament. This state of affairs developed during the period of four months following the passage of an ordinary hydatidiform mole. On admission the uterus was the size of a 4 months' pregnancy and showed a marked bulge into the region of the left broad ligament, where a peculiar crepitant feeling could be felt on bimanual examination. The pregnancy test was positive at 1/50 dilution. Pan-hysterectomy was carried out at once and at the point where the crepitation had been felt the mole was found to have burst through into the space between the layers of the left broad ligament. A few vesicles had actually penetrated into the peritoneal cavity and were lying there with a small amount of free blood. Sections showed molar tissue in varying stages of activity penetrating deeply into the uterine muscle. In one place a mass of trophoblast was found within a blood vessel in the wall of the uterus (Fig. 5). The pregnancy test became negative exactly one month after the operation and the patient has remained well for over two and a half years.

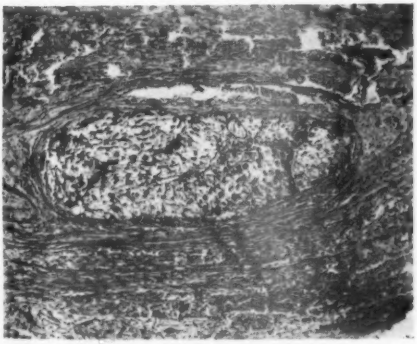

FIG. 5.—Section from an invasive mole (Path. No. 14707) which perforated right through into the broad ligament, showing a large mass of trophoblast situated within a blood vessel in the uterine wall.

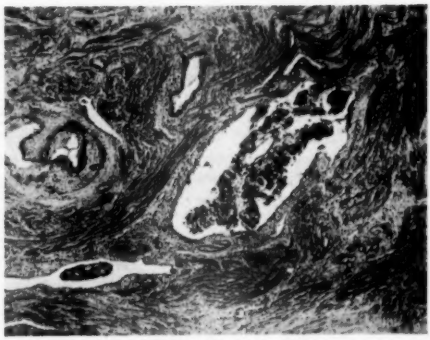

FIG. 6.—Example of an invasive mole with progressive changes (Path. No. 6901). A well-marked portion of cellular and proliferative trophoblast is seen within a blood vessel.

Case No. 332/51 (Path. No. 6901). In this final example a condition bordering upon malignancy was found. The patient, a 4-para aged 29, was admitted with a very large mole and the pregnancy test was positive at a dilution of 1/100. The mole was removed by hysterotomy on account of its size and the very strongly positive hormone reaction. The uterine cavity was carefully inspected and palpated and no suspicious areas could be found. But the pregnancy test remained strongly positive and was still present at 1/100 dilution one month after the operation. For this reason pan-hysterectomy was carried out on the thirty-third day after the removal of the mole. Two dark nodules of growth were found within the muscle of the posterior wall of the uterus. Sections showed well-formed molar tissue and masses of actively proliferating trophoblastic cells penetrating the wall of the uterus. Many of these infiltrations were found within the lumina of the blood vessels, and there seemed little chance that trophoblastic tissue in this favourable situation would be likely to undergo regressive changes (Fig. 6). In many areas the appearance was strongly suspicious of malignancy, and there can be little doubt that the picture would shortly have become frankly malignant if the uterus had not been removed. This patient was alive and well four and a half years later.

Conclusion

A true perspective of these cases can only be gained by a combined study of the clinical findings, the hormonal reactions and the results of the pathological investigations of all tissues removed.

Uterine enlargement which persists after the evacuation of a mole, or which increases progressively in size, or becomes irregular is of great significance, as also is the persistence of uterine bleeding or the recurrence of irregular bleeding after it has once ceased. The appearance of vaginal metastases is sometimes the first sign of the development of malignancy, although even then it is not always a proof of chorion-epithelioma, as in a few instances these deposits may result from the invasive form of mole. Pulmonary metastases similarly develop very early and hæmoptysis should always be carefully watched for and routine chest films should be called for in all borderline cases.

The gonadotrophic hormone of the urine is probably the most reliable single guide to the progress of the case. The persistence of a positive pregnancy test for a period longer than one month after the evacuation of the mole is looked upon with the greatest suspicion, particularly if the titre is steadily increasing. The recurrence of a positive test after a free interval of varying length is also highly suspicious, it being always remembered that normal pregnancy is to be excluded. In a few cases, too, the presence of luteal cysts is associated with a positive reaction. But if these things can be excluded the persistence or the return of a positive reaction is usually due to the presence of a residual mole or an invasive mole or to the actual development of chorion-epithelioma.

The importance of the routine pathological examination of all tissues removed cannot be stressed too strongly. Curettage sometimes gives information which clinches the diagnosis of malignancy, especially when taken in conjunction with the clinical findings and the results of the hormonal assay. More often than not, however, the complete pathological picture can only be obtained after a full examination of the uterus, if the removal of this organ is justified, and this not infrequently involves the sacrifice of the specimen in order to obtain serial sections of the suspicious areas.

Finally, in reaching a decision as to the treatment of borderline cases it is sometimes helpful to bear in mind the percentage chance of the occurrence of malignancy. In these 64 cases of mole 78·1% proved to be simple uncomplicated cases who remained well afterwards. The incidence of chorion-epithelioma was 9·4%, leaving a borderline group of 12·5% of cases who developed conditions varying from a simple invasive mole to conditions scarcely distinguishable from actual malignancy. In all of these borderline cases hysterectomy was called for and was carried out. In only two cases of this series was it found that there was no significant pathological condition after hysterectomy had been performed, and these patients were classified as uncomplicated moles. In view of the risks involved a decision as to treatment must be made early enough to save the patient, and must at the same time avoid, if possible, the unnecessary sacrifice of the uterus. But in cases where there is any reasonable doubt it would appear to be better to remove the uterus, in view of the known risks of malignancy, than to regret the lost opportunity if the patient should subsequently develop chorion-epithelioma.

Acknowledgment

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Section of General Practice

President—GEOFFREY BARBER, O.B.E., M.A., M.B.

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Bread and Tears—Naughtiness, Depression and Fits Due to Wheat Sensitivity

By GUY DAYNES, M.R.C.S., L.R.C.P., D.C.H.

WE would never punish a child for something we knew he could not help doing, but what if we thought the child *could* help it? Just for a moment imagine you were Anna's parents, and think what you would have done—here is her story:

Case I.—Anna M., aged 2. She had severe acute tonsillitis and otitis media when she was 11 months old. From then on she had woken at night with two or more crying attacks lasting over a period of some twelve months. Often she seemed awake but confused not knowing anybody and would sob until she went off to sleep again. The more severe attacks would be ushered in with a piercing scream. While in her pram, her mother described her as having a "far away look" at times. She always "behaved badly" and did not play well with other children, being spiteful to them. She was pale and had a poor and unpredictable appetite. Her motions varied; sometimes they were loose and yellowish with a very offensive smell, at other times they were normal. Examination revealed little else beyond a pale child who was thin, and perhaps the absence of fat was most marked in the buttocks. She was placed on a gluten-free diet, which she started on a Friday. She had a bad screaming attack that night, but on Saturday night was "grizzly only". On Sunday night she slept the whole way through the night without waking for the first time for a year. In the next two weeks she had two screaming attacks, each of which had been preceded by an accidental lapse in diet. On one occasion a well-meaning friend had given her a proprietary ice cream and on the other the dog had stolen some bread put out for the rabbits, had taken it on to the lawn where he and Anna had shared it. When she was next seen, she appeared much happier, behaved well, was eating well and had gained weight. The other children had all commented on how much nicer Anna was now. She has remained well, but lapses of diet still adversely affect her.

Study of over 40 similar cases has led me to formulate a syndrome which has been called the pre-cœliac syndrome. Typically a child between 1 and 5 years becomes naughty and difficult a few days after the onset of an acute infectious illness such as measles or gastro-enteritis. He is irritable, negativistic and spiteful, sleep is disturbed and he wakes up many times in the night and often screams; his appetite is poor, he fails to gain weight, his abdomen is often distended and the stools may become bulky, pale and offensive. This condition, if left untreated, usually rights itself after a month or two, but may last for much longer, in which case slight *petit-mal*-like attacks may develop, in addition to worsening of the other symptoms. I have been placing these children on a gluten-free diet at the earliest opportunity, and the symptoms respond dramatically, usually within two or three days. They relapse if a premature return to normal diet is made.

Here are two more examples of the syndrome:

Case II.—Peter B., aged 4. At 20 months he had gastro-enteritis after which his mother noticed progressive deterioration in his behaviour, and he became wet and dirty day and night, when previously he had been dry and clean. A month later he started to have "blank turns". I thought that these were *petit-mal* attacks and treated him with sedatives. At this time he had "night terrors" and slept badly. There followed a period of improvement lasting about three months until he had an attack of influenza. After this he became much naughtier and completely uncontrolled, being extremely difficult to examine—in fact "a horrid child". He began to have pale stools and his weight remained stationary. When he was 2¼ he had another attack of diarrhœa and vomiting and deteriorated still more. The attacks were more frequent and no longer influenced by sedatives, notwithstanding a doubled dose, and his behaviour became stranger still. Transferring him from the nursery to his high chair in the dining room nearly always brought on an attack unless his attention was held by someone playing with him. The attack would start by him becoming stock still and staring in front of him, sometimes for as long as a minute; then his head would twitch, there was a fluttering of his eyes and a spasmodic movement chiefly of the upper limbs, and he would often "pant". During this time there was a rush of words and sentences, generally lacking coherence, and often ending with a scream. The attacks were worse following any excitement during the previous twenty-four hours.

A further curious symptom was a dryness of the mouth, the child seeming to have difficulty in eating anything other than moist foods. His appetite was very poor and highly capricious. His play was characterized by over-activity, charging about the nursery, often knocking into furniture and indeed the wall, so much so that anything breakable had to be removed from the room. His speech at this time was most odd; he talked very slowly with an exaggerated drawl so that some words seemed as though they would never stop. His gait was strange, and he was wobbly if made to stand still. An EEG was abnormal by reason of excess high voltage fast rhythm, but there was no epileptic activity seen, the pattern suggesting "a diffuse organic process". I considered the possibility of cœliac disease and I put him on a gluten-free diet.

The response was dramatic—his behaviour improved, and the attacks disappeared. Sedation was discontinued. Three weeks later, he became naughty again, the trouble was traced to some flour-containing blancmange. Removal of this from the diet restored him quickly to normal. He has never really looked back since then, and now is a charming little boy, apparently normal in every way. A more recent EEG showed much less fast activity than in the first record, but was still abnormal by reason of definite alpha activity in the left occipitotemporal areas. In April 1955 he relapsed following the eating of chocolate for a week or so—so he must stick to his gluten-free diet.

Case III.—Frances W., aged 2. She had a four months' history of naughtiness, screaming at night, loss of appetite and failure to gain weight following gastro-enteritis. Her stools were pale and foul-smelling and her abdomen was prominent and buttocks small. There was a steady response to a gluten-free diet with sharp relapse of symptoms when returning to normal diet. She had to remain off gluten for four months but then was able to return to normal diet without relapse.

The diagnosis of pre-cœliac syndrome is easy. The main symptoms (Table I) were shown in the 3 cases described.

TABLE I.—SYMPTOM ANALYSIS OF PRE-CŒLIAC SYNDROME

		%
1.	Child becomes naughty, irritable and "difficult"	90
2.	Stools are bulky, pale and offensive	90
3.	Disturbed sleep and screaming at night	85
4.	Poor appetite with tendency to abdominal pain	80
5.	Failure to gain weight	50
6.	Prominent abdomen and small buttocks	30
7.	*Petit-mal*-like attacks	25
8.	Skin rashes and skin irritation	10
9.	Abdominal pain with nausea	10

Sleep disturbance is a common presenting symptom. Abdominal pain mainly seems to be due to distension of the small bowel with gas. The naughtiness is characterized by being the sort that other children dislike, readily distinguishable from the sort of naughtiness which makes other children heroes of the kindergarten.

There is a very real chance of a child being punished for naughtiness; this may not be the only reason why a child is wrongly punished on account of bread being eaten at an unfortunate time, for adults may get a somewhat similar trouble after infective illnesses, causing them among other things to be highly irritable.

Case IV.—Mr. S., aged 41. A week after influenza this man became very irritable and depressed with a headache and some diarrhœa. I regarded him as an early case of "headache/insomnia/depression (H.I.D.) syndrome" (the name I have given to the adult condition). He was placed on a gluten-free diet with a complete remission of symptoms twenty-four hours later. It was then that he confided to me that on the day the condition developed he had punished his son far more severely than the occasion justified, and looking back he was sure that his own mental state was the reason.

"Headache insomnia depression syndrome" in adults is as readily recognizable as is pre-cœliac syndrome in children.

Typically, two to fourteen days after the onset of an acute infection, such as influenza or gastro-enteritis, the patient develops a headache, starts being wakeful at night and becomes progressively depressed. Abdominal distension and diarrhœa may occur and are often associated with pale, offensive stools; pruritus ani may be a prominent feature. A gluten-free diet brings prompt relief of all symptoms.

Case V.—Mr. L., aged 48. Had just returned from a four months' visit to Damascus where the insanitary cooking conditions predisposed to four or five infective diarrhœa attacks. He was passing bulky, pale stinking motions at frequent intervals, and had considerable excoriation and inflammation of the skin around the anus. In addition he was feeling in "rather poor spirits" and was "edgy and difficult" at the office and prone to headaches. He was treated with a gluten-free diet, which caused a remission of his symptoms, the nervous ones being the first to improve. He stuck to the diet rigidly for the first four weeks but "let up" a bit after that. This back-sliding promptly resulted in a deterioration in the stools with a severe recurrence of his anal skin soreness. These quickly got better again once he kept rigidly to his diet and he has had to remain on it.

Case VI.—Mrs. M., aged 44. Developed "blinding headaches" with insomnia, depression and pruritus ani following a severe virus infection associated with meningism—she gave a history of similar symptoms following dysentery in India on many occasions. Her symptoms responded promptly to a gluten-free diet, but extreme sensitivity remained. One chocolate, a piece of "cœliac bread" or a small glass of whisky was sufficient to reproduce the headache lasting for about two days.

Comparisons.—Two syndromes have been described both of which appear to be cured by a gluten-free diet. Nevertheless, one cannot conclude that because the gluten-free diet seems to effect a cure, the condition is caused by gluten in bread. Such additional evidence as is available must be examined before drawing such a conclusion.

Does not the very close similarity justify pre-cœliac syndrome being regarded as the human counterpart of canine hysteria? (Tables II and III).

TABLE II.—COMPARISON BETWEEN PRE-CŒLIAC SYNDROME AND CANINE HYSTERIA

Canine Hysteria	*Pre-cœliac Syndrome*
Onset often associated with infection.	Onset usually associated with infection.
Unfriendly and frightened.	Unfriendly and frightened.
Will bite the hand that feeds it.	Spiteful and kicks mother and doctor.
Has very disturbed nights.	Has very disturbed nights.
Abnormal slow unsteady walking.	Abnormal slow staggering gait.
Has dry mouth.	Has dry mouth.
Has characteristic fits.	Has similar fits (*see* Table III).
Has pale, bulky, foul-smelling stools in later stages of the disease.	Has pale, bulky, foul-smelling stools frequently.
Has severe skin irritation and scratches a lot.	Many cases have skin irritation with or without rash.
Most cases respond promptly to diet which excludes chemically treated flour.	Most cases respond promptly to gluten-free diet.

TABLE III.—COMPARISON OF FITS IN CANINE HYSTERIA WITH THOSE IN PRE-CŒLIAC SYNDROME (*See* CASE II.)

Canine Hysteria	*Pre-cœliac Syndrome*
Attacks induced by nervous strain or change of environment.	Attacks induced by nervous strain or change of room.
Frightened look at beginning of fit.	Frightened look at beginning of fit.
Sits in sphinx-like posture at beginning of fit.	Sits stock still and gazes into space at beginning of fit.
Jerks head backwards at beginning of fit.	Jerks head backwards at beginning of fit.
Runs round cage barking furiously.	Runs round room shouting or screaming.
Knocks food and water dishes over and gets cage into filthy mess.	Knocks into walls apparently without seeing them.
When running stops, staggers round like drunken person.	When walking, has staggering drunken gait.
Often shakes itself at end of fit.	Often shakes himself at end of fit.
Looks very miserable at end of fit.	Looks very miserable at end of fit and often cries.
Fits stop quickly when chemically treated flour is withdrawn from the diet.	Gluten-free diet stops fits quickly.
The longer they have had fits, the longer they take to recover fully on diet free of chemically treated flour.	The longer they have had fits, the longer they take to recover fully on gluten-free diet.

(The description of canine hysteria fits is from Mellanby, 1946.)

Discussion.—Clearly there is a basic similarity of disease pattern between pre-cœliac syndrome, H.I.D. syndrome, cœliac disease and canine hysteria. Not only is the symptomatology similar but also they all respond to the withdrawal of wheat flour from the diet. Whereas cœliac disease remains relatively rare, I contend that pre-cœliac syndrome and H.I.D. syndrome are very common conditions. I have been able to diagnose some 50 or 60 cases in the past two-and-a-half years in a private general practice with two infant welfare clinics per week.

I believe that these syndromes are the result of a sensitivity to a digestion product of wheat flour, at a time when patients are specially liable to develop this, i.e. when they have an infection that can cause cell damage. Some may think these changes I have described are the result of direct toxicity by the pathogen following the illness. I do not think this is true because I have cases where the preceding cell damage has been caused by trauma

(concussion) and not by any infective process at all. Belief in my explanation of these cases demands acceptance of two fundamental concepts:

(1) That damage to nerve tissue can be followed by sensitization to certain common allergens.

(2) That such sensitization once caused can be temporary only provided the allergen is withdrawn early.

Allergists have described cases such as mine in the past, but unfortunately scant attention has been paid to their observations by the rest of the profession (Rowe, 1944; Walker, 1954).

It is quite true that the majority of cases get better anyway, but they often take quite a long time to do it—weeks, or even months elapse before cure results. All are very glad when it is over, and anyone who can cut short this miserable convalescent period is sure of gratitude. Quite apart from this, permanent damage to the brain cells may be left after some of the longer illnesses, so that treatment must be started before the "point of no return" is reached.

Falconer and his colleagues (1955) found such damaged areas at operation in some of their cases suffering from temporal-lobe epilepsy and many of their cases had a preceding history of infection followed by personality changes similar to my cases. May not some cases of temporal-lobe epilepsy be end-results of these syndromes when the damage has been severe and mainly cerebral, just as cases of cœliac disease may be end-products when the damage has been mainly intestinal? Should we not re-examine our cases of *petit-mal* and behaviour and sleep disorders in children, also cases of reactive depression? Cases of recurrent headaches and insomnia might also be reviewed; in them you may find one or other of these syndromes, and have the satisfaction of being able to put them right. Whatever the significance of my findings may be, it is certain that at least we have in the gluten-free diet a fine symptomatic treatment.

Finally, I must face the most difficult problem of all. Bread and other flour products have formed the staple diet of the inhabitants of Europe and America for centuries. Is it possible that this common article of diet has been acting in the way I have suggested without anybody recognizing it, or is there some factor which has made these disorders much more common in recent years? Certainly nervous disorders in general seem to be increasing considerably in most strata of society at the present time. Ever since Mellanby (1946) showed a link between canine hysteria and agenized wheat-flour many people have been wondering whether the chemical treatment of flour has been adversely affecting the health of the population. Nevertheless, I do *not* think that agene alone is responsible for these syndromes, nor incidentally do I accept the current view that it is the sole cause of canine hysteria.

I have observed in many of my cases that the severest reactions have been precipitated by *stale* wheat flour. This suggests that the basic toxic allergen may be found in the staling process of flour itself and flour products. We know that vitamin E is progressively destroyed during the staling process. When flour is treated by agene or chlorine dioxide, vitamin E is destroyed more quickly and completely (Horder *et al.*, 1954). Let our chemical research workers find the cause of its destruction, and our offending allergen may be revealed. At the moment this remains but an idea, but further research will find the truth, and when it is found many another door may open.

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Section of Orthopædics

President—Sir REGINALD WATSON-JONES, M.Ch.Orth., F.R.C.S.

[*October* 4, 1955]

[Continued from April *Proceedings*, p. 184]

Rupture of the Lateral Ligament of the Ankle with Interposition in the Subtaloid Joint.—MICHAEL J. COX, M.B.

Mrs. A. M., aged 24.

On 21.9.55 she stepped on a bottle top which threw her over and she suffered a severe inversion injury of the right ankle. She was admitted to the Orthopædic and Accident Department, The London Hospital.

On examination soon after the injury the foot was held rigidly in almost full inversion. This inversion could not be corrected passively. X-ray examination revealed no bony injury or tilting of the talus. An examination was then made under general anæsthesia to see if the inversion was due to muscle spasm or mechanical block. The foot could only be brought to neutral with great difficulty but this position could not be maintained. X-ray in forced inversion showed marked tilting of the talus.

On 26.9.55 an operation was performed by Mr. C. Attenborough, and complete rupture of the lateral ligament was found.

The anterior fasciculus was torn from the lateral malleolus, the middle fasciculus from the calcaneus and the posterior fasciculus from the lateral malleolus.

The middle fasciculus was rolled into the subtaloid joint, when this was removed full eversion was easily obtained. The ligaments were then reconstructed. A pressure dressing and backslab were then applied for two weeks and a below-knee plaster for two months.

Comment.—This is believed to be the first recorded case of interposition of part of the lateral ligament of the ankle into the subtaloid joint giving rise to persistent mechanical block to reduction of the deformity.

The discussion was concerned with the length of immobilization necessary to ensure healing of torn ligaments. The general consensus of opinion was that six weeks was sufficient provided that apposition of the frayed edges was ensured either by manipulation or open repair.

[*December* 6, 1955]

Congenital Postural Scoliosis

By DENIS BROWNE, F.R.C.S.

Pathology.—The name of the condition described in this paper indicates that the scoliosis is present at birth and that it consists in the persistence of a posture affecting a normal spine. The posture must have been abnormal before birth with the child lying bent to one side (Fig. 1). It is my opinion that the obstinate and crippling condition known as "idiopathic scoliosis" originates, certainly in the majority of cases and possibly in all, as this sideways bend *in utero*. The reasons this hypothesis is not generally accepted are: firstly, the curious omission in orthodox orthopædic teaching of any study of what may be called the "orthopædics of the fœtus" (Browne, 1936); and secondly, the extremely inconspicuous nature of the deformity in its early stages. In the first months of life, when it is essential for a rapid and complete cure that the diagnosis should be made, all that is noticeable is not a positive abnormality, since the position of the spine is one which any onrmal baby can, and frequently does, assume: there is simply the absence of the power of assuming the reverse curve of the spine.

In consequence of the lack of understanding of the origin of the deformity, both undue optimism and undue pessimism have arisen. The optimism is because any baby can assume such a curve of the spine and have it recorded in an X-ray; when the spine is in later life

found to be straight it is assumed that there was a congenital scoliosis, and that this has righted itself spontaneously. The pessimistic outlook, that idiopathic scoliosis is a disastrous progressive condition, resistant to all forms of treatment, comes from lack of recognition of the early curable stages and also from the lack of any device to reverse and control the curve.

The curve is nearly always concave to the right, which might be expected from the usual preponderance of muscular strength on that side; and the muscles on the concave side have in their shortened state a considerable mechanical and physiological advantage in pull over those which are stretched on the convex side. When the body weight is added to this advantage as the child sits up and walks the curve tends inevitably to increase, and as it increases so does the advantage of the one side over the other. The result is the well-known progressive and accelerating deformity which ends in an extreme scoliosis which not only cripples the patient but also markedly shortens the expectation of life owing to the diminution of the vital space in the chest.

In addition to the factors described I think that it is highly probable, though it cannot be proved, that there is a factor which I would call "pressure dysplasia". By this I mean an interference with growth and with the power of subsequent growth of bones and joints caused by abnormal forces *in utero*. I consider that it is this pressure dysplasia on the lower end of the tibia and the inner side of the ankle which causes the notorious tendency of club-feet which have had the deformity thoroughly corrected to grow again into the original deformity; the outer side of the foot and ankle always growing better than the inner side. When the spine has developed before birth in a scoliosis there must be more pressure on the edges of the vertebræ on the concave side than on the convexity; and in consequence a varying degree of pressure dysplasia seems to me extremely probable. If it did occur there would be a differential rate of growth on the two sides which would reinforce the factors making for increase of the deformity which have already been mentioned (Fig. 2).

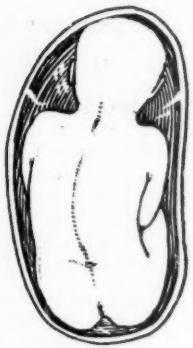

FIG. 1.—Hypothetical position of fœtus in utero causing congenital postural scoliosis.

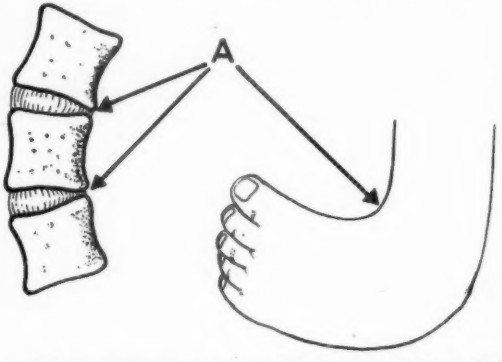

FIG. 2.—Diagram showing points (marked "A") at which "pressure dysplasia" is assumed to occur in talipes and in congenital postural scoliosis.

Diagnosis.—To define congenital postural scoliosis it is necessary to set out the related but different conditions with which it may be confused. It is not:

(1) An actual malformation of the bones of the spine, such as a wedge vertebra or spina bifida occulta. The bends due to these conditions have a quite different prognosis, and are not progressive in the same disastrous way.

(2) A collapse of the spine coming on in childhood, due to actual weakness of the bony substance, as in cases of osteogenesis imperfecta or hypophosphatasia.

(3) As already mentioned, it is particularly not the record on an X-ray film of the wriggling and squirming in which most babies indulge, especially when held down on a hard table.

The diagnosis of this condition is not primarily a matter of X-rays, though these are necessary for recording purposes; it is a matter of observation. If the baby is placed prone and stimulated in various ways, it will be noticed that though he will turn freely to one side, no attractions or blandishments will get him to curve his spine into the corresponding position on the other side. On manipulation a very distinct difference will be found between the ease of forced bending to right or left.

In addition, there is a valuable sign which can be found in many cases; that of transverse wrinkling of the skin on the concave side, the wrinkles resembling those at the corners of adult eyes (Figs. 3 and 5).

FIG. 3.—Newborn baby lying in typical position. Note the skin creases.

FIG. 4.—Splint recommended for early treatment of these cases.

FIG. 5.—Child of 8 months in typical position.

FIG. 6.—Child of 4 years with "idiopathic scoliosis" in progressive stage.

FIG. 7.—Child of 15 years with "idiopathic scoliosis" which has been steadily developing.

Prognosis.—My own opinion is that the condition I have described probably never corrects itself, and that if the child is allowed to walk with the scoliosis uncorrected the curve inevitably increases in the way stated. I have no actual evidence on this point, as I have never considered it morally justifiable to let an infant in the curable stage progress to life-long crippling simply for the sake of what in any event would be an inconclusive observation. I have, however, records of 12 cases of typical idiopathic scoliosis in which the mothers had noticed the curve before the child walked, and I regret to say that in most of their histories there was some variation on the complaint: "I kept telling them the baby's spine was crooked, and they just laughed at me". This absence of recognition of the danger of an apparently trifling condition, however regrettable, is hardly surprising in view of the complete lack of warning in standard text-books on orthopædics, not to consider general medical education.

As to its curability, I have now 32 cases in which the condition was found and treated before walking began, and in none of them has "idiopathic scoliosis" developed. It seems to me that the condition has many points of resemblance to congenital dislocation of the hip, in which complete correction is easy to obtain if effective treatment is started before walking has begun, and in which a really satisfactory result is impossible after a very early age.

Treatment.—In treating congenital postural scoliosis, as well as talipes and congenital dislocation of the hip, the principle of "controlled movement" is of the utmost importance. This consists in cutting out by various devices the movements which tend to maintain or increase the deformity, while allowing and encouraging those which do no harm, or which correct it. My splints for talipes and congenital dislocation of the hip work on this principle, and I have devised one on the same lines which is very effective in these infantile scolioses. It is unfortunate that at the present time the classical techniques of correction of these deformities consist almost entirely of immobilization, which has no effect on the disturbance of muscle balance, removes the indispensable stimulus of movement to correct growth, and, in addition, atrophies the muscles and stiffens the joints. At one time I used plaster-of-Paris beds for treating these cases, but they were extremely difficult to make effectively, had the well-known disadvantages of this rough, heavy, fragile and absorbent material, and the child grew out of them very rapidly.

The splint illustrated (Fig. 4) is made of sheet aluminium, covered with sponge rubber. The child is held in it by putting his arm through a hole in the curved upright wall, and then pulling his spine against it into a curve the reverse of the original one by means of a broad padded strap. He can perform almost any movement of the muscles and joints; the one thing he cannot do is to get his spine back to its primitive scoliosis. He is kept in this apparatus continuously, being taken out only for washing and manipulations and exercises. These exercises, when carried out under skilled instruction, are of very great value. Only when the child will turn as readily and as far to one side as the other, and when the spine appears quite straight on sitting up, is he allowed to take the erect position and learn to walk. For some time after he is let free in the day-time he is returned to the splint to sleep; it is much better to keep him in it too long rather than to let him out too soon.

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Differential Diagnosis of Infantile Scoliosis

By J. C. SCOTT, M.A., M.D., M.S., F.R.C.S.

Nuffield Orthopædic Centre, Oxford

INFANTILE scoliosis, by definition, is one which is present at or before the age of 2 years. Scoliosis before the age of 2 may be a very disturbing feature, as far as the parents are concerned, and the ability to give a reasonably accurate prognosis is of considerable importance—particularly if it is possible to say from the type of curve that the outlook is good.

The types of scoliosis in this group are:

(1) *Postural.*—To assess the frequency and importance of this condition I have—through the courtesy of the Department of Social Medicine, University of Oxford, and Dr. Kemp, their radiologist—reviewed the X-rays of some 75 normal children who were X-rayed soon after birth, and at regular intervals after that. The Group that I examined were all now 2 or more years of age.

X-rays at 6 months show a lateral curve of 10 degrees or more in 80%, at 12 months in 46%, and at 18 months in 7%. There was none with lateral curvature at the age of 2. This sequence of events might reasonably be interpreted as indicating that the infantile

FIG. 1.

FIG. 2.

FIGS. 1 and 2.—Postural scoliosis at 6/12 and 2 years.

FIG. 3.

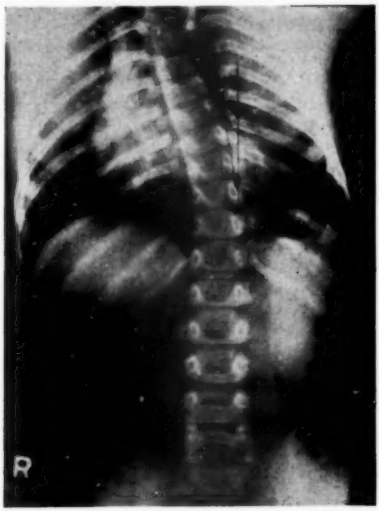

FIG. 4.

FIGS. 3 and 4.—Examples of resolving infantile scoliosis—curves disappeared without treatment.

alignment of the spine persists until the erect posture is assumed, and that it is this that stimulates the disappearance of the postural lateral curve (Figs. 1 and 2).

(2) *Resolving*.—This differs from the above in that it persists after standing—also the curve tends to be greater, and there is some rotation present (Figs. 3 and 4). These recover fully without treatment, but may persist up to 4 or 5 years of age. They are uncommon, but a number have been reported by Harrenstein (1936), and Scott and Morgan (1955), and there are probably quite a few which pass unrecognized.

FIG. 5.

FIG. 6.

FIGS. 5 and 6.—Progressive infantile scoliosis at age 4 years and 12 years.

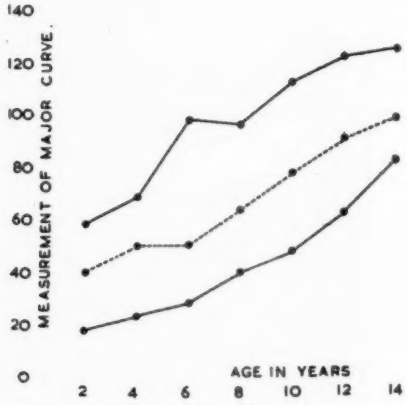

FIG. 7.—Shows minimum, maximum and average in 28 patients with progressive infantile scoliosis.

FIG. 8.—Congenital scoliosis.

(3) *Progressive infantile idiopathic.*—This may be indistinguishable from Group 2 up to 2 years of age, and difficult even up to 3 or 4 years. The curves are in the dorsal spine. They are shorter and more severe, both in the lateral curve and in apical rotation, and the apex is to the left (Figs. 5, 6, 7). These usually begin to increase at an early age.

(4) *Congenital.*—With hemivertebræ (Fig. 8), and usually diagnosed later in life. In a series of approximately 50 patients with congenital scoliosis it was found that the average age of diagnosis was 11. There are two types—those in which the abnormalities are all

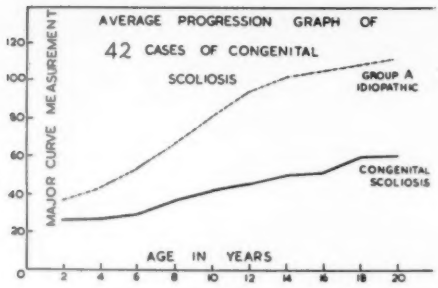

FIG. 9.

on one side, and often localized to the upper dorsal region (Fig. 8)—and those in which hemivertebræ are present on both sides, and in which there is little clinical evidence of abnormality. X-ray readily distinguishes these from the others. Deformity in both these types is usually relatively mild compared with (3) (Fig. 9), and the prognosis is relatively good. In spite of this it should be remembered that paraplegia, the rare complication of scoliosis, occurs more frequently in congenital than in any other type of scoliosis.

(5) Very few lateral curves of known ætiology appear before the age of 2.

A diagnostic label is a guide to prognosis, and also with regard to the ætiology. The first Group is of little importance, as far as this aspect of the problem is concerned—except to indicate how postural influences affect development of the normal spinal alignment. In the second Group it is of particular interest that a curve may be present, measuring 20 degrees, and involving several vertebræ, but without containing that progressive element which we have always connected with vertebral growth, and about which we know so little, and may then spontaneously disappear. The third type is, of course, the most serious and the one about which—from the point of view of treatment—we most urgently need knowledge of the ætiology.

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Lumbo-sacral Fusion for Low Back Pain

By ERIC SHAW, F.R.C.S.Ed.

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(*From the Nuffield Department of Orthopædic Surgery, Oxford*)

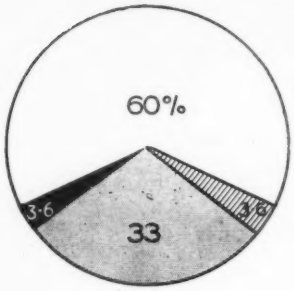
THIS paper is based on a follow-up review that Mr. J. G. Taylor and I did when we were at the Wingfield Morris Orthopædic Hospital. We were impressed by the good *immediate* results of lumbo-sacral fusion in the treatment of chronic low back pain, and we wondered why it was not done more readily. It was the apparent reluctance to use this form of treatment that stimulated us to investigate the results.

The records of 61 patients between 1934 and 1954 were available, and it was possible to follow up 52 of these. This represented 55 operations which were all fundamentally the same—onlay grafts using autogenous bone—and they were undertaken for the treatment of chronic low back pain due to a non-infective structural weakness.

The review was both clinical and radiological, all the patients being interviewed and examined by one of us, but estimation of bony fusion or otherwise was solely by study of flexion-extension lateral and serial antero-posterior radiographs. In the serial antero-posterior views the points sought in confirmation of bony union were moulding of the grafts in response to stresses and strains, thickening of the grafts, and lastly a natural conformity of the graft bone with the appearance of the host bone in the area so that it no longer looks like a foreign body.

The fusion rate shown by this method of investigation is illustrated in Table I.

TABLE I.—OPERATIVE RESULTS



Successful ..	33
Doubtful ..	2
Failed	18
Died	2
Total operations	55

TABLE II.—COMBINED TABLE

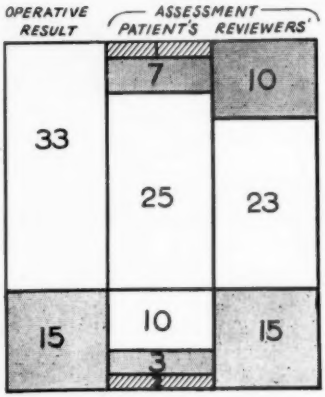


TABLE III.—CORRELATION OF OPERATIVE RESULTS WITH DISEASE PROCESS

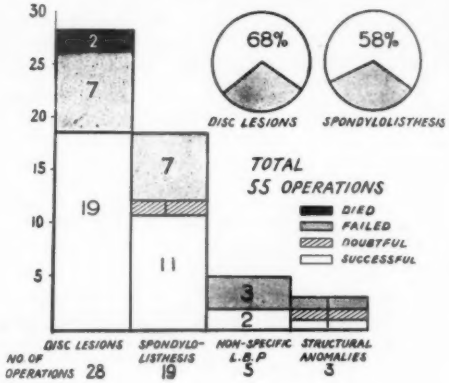


TABLE IV.—CORRELATION OF OPERATIVE RESULTS WITH GRAFTING TECHNIQUE

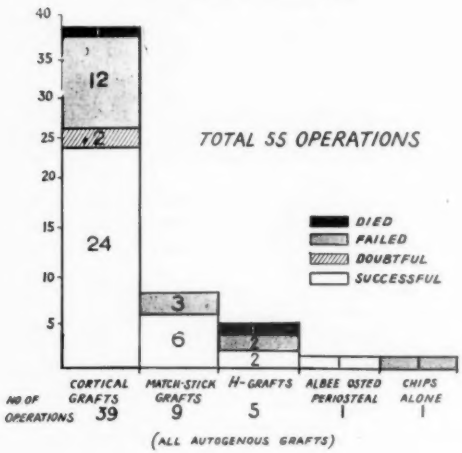


TABLE V.—RELATIONSHIP OF OPERATIVE RESULTS TO PERIOD OF PLASTER BED IMMOBILIZATION

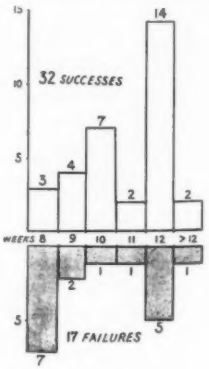


FIG. 1A.—(*Case I*).

FIG. 1B.—(*Case I*).

FIG. 2A.—(*Case II*).

FIG. 2B.—(*Case II*).

Each patient was questioned regarding any remaining symptoms and about his ability to work and play. He was asked his own estimation of whether his operation had been worth while. 73% of patients stated that they were satisfied. Table II tabulates the successes and failures from the operative point of view and that of the patients and reviewers. It can be seen that the figure of 35 satisfied patients (73%) was made up from 25 with solid fusions and 10 in which fusion had failed. Presumably these latter 10 had fibrous unions firm enough to control their pain.

FIG. 3A.—(*Case III*).

FIG. 3B.—(*Case III*).

FIG. 4.—(*Case III*).

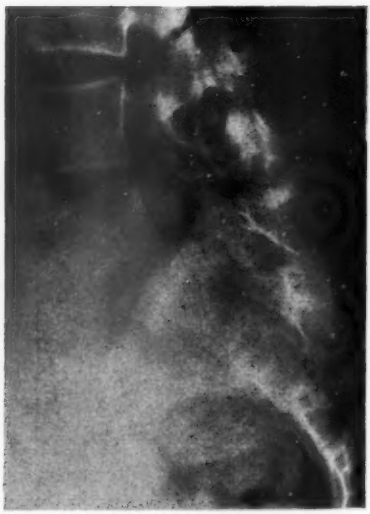


FIG. 5.—(*Case III*).

It will also be seen from Table II that 8 patients who had solid fusions were not pleased with the result symptomatically. In 7 of these 8 cases we think a reason can be found why operation failed to relieve the patient of symptoms. Three of them had a generalized type of spondylosis and there was evidence of it above the level of the graft. One patient had a pseudarthrosis at the upper end of the graft which was unnecessarily long. In one patient who had had a transitional vertebra fused to the sacrum we felt that his symptoms had been, and still were, due to a disc degeneration in the space above the transitional vertebra. One patient with a successful lumbo-sacral fusion appeared to have a

sacro-iliac arthritis. One patient seemed to be an hysteric. In the eighth patient we could not find any reason why successful fusion had not relieved her symptoms.

In all these cases where a successful fusion had failed to relieve symptoms the operation had been done for disc degeneration. When done for spondylolisthesis a solid fusion was always successful in relieving symptoms.

In an attempt to analyse the causes of failure to obtain fusion the operative results were correlated with the disease process for which it was undertaken (Table III), but this analysis was not helpful. However it did bear out the clinical impression that it is easier to obtain fusion in disc lesions than in spondylolistheses. 68% of disc lesions obtained fusion compared with 58% in spondylolisthesis.

Table IV correlates the operative result with the type of bone graft used. Excluding two doubtful cases and one death the failure rate with solid cortical grafts and match-stick grafts was the same in each, that is one-third.

Table V correlates the length of post-operative plaster-bed immobilization with the operative result, in those cases where this time was known.

It can be seen that of 17 failures 7 were immobilized for only eight weeks. On the other hand of 32 successes only 3 were immobilized for so short a time. Another 2 were immobilized for only nine weeks. Ten to twelve weeks seems to be the optimum time in these cases where internal fixation is not used. Other possible causes of failure may have been the use of an insufficient quantity of bone and poor apposition of the graft to its bed (Fig. 1A and B).

Summary.—The failures fall into 2 categories: (I) Failure of fusion and (II) failures of selection.

I.—(1) The use of insufficient bone. (2) Poor apposition of the graft to its bed. (3) Inadequate immobilization. (4) Attempts to span too many vertebræ (in this series often resulting in a painful pseudarthrosis at the upper end of the graft).

II.—(1) Fusion of a local area in a diffuse pathology (where spondylosis is generalized). (2) Fusion of the wrong area (e.g. lumbo-sacral fusion for sacro-iliac arthritis). (3) Fusion in the wrong patient (e.g. hysterics).

Illustrative cases.—I.—E. D., a woman of 30 had had severe low back pain for six months. Her occupation was a strenuous one (nursing) and she lived abroad. She had a spondylolisthesis of L5 on the sacrum. Fusion was advised and at operation twin cortical grafts were laid from the spine of L4 to the sacrum. Fig. 1A shows that the grafts are not in close contact with the spines; and Fig. 1B shows them standing well away from the laminæ. At follow-up one year later X-rays show free movement between the spines of L4, 5 and S1 and between the spines and the grafts. This patient was only doubtfully satisfied and was classed as a failure by the reviewers.

II.—M. E., a man of 42 years, had had low back pain for three years. He had a chronic disc degeneration between L4 and a transitional vertebra. Twin grafts were placed, as is seen in Fig. 2A and B, from the spine of L2 to the sacrum. These X-rays taken at follow-up two years later show no movement between the graft and the spines of L4, the transitional and the sacrum which is the area required to be fixed; however, there is movement above the level of L4. In this case the patient was cured of his pain but we believe these unnecessarily long grafts are sometimes the cause of a "different pain" at the site of the pseudarthrosis.

III.—P. B., a girl of 10 years, presented as a case of bad posture. She was found to have a spondylolisthesis of L5 on the sacrum. There was a neural arch defect in L5 but no other defect could be seen in the flexion-extension X-rays taken before operation (Fig. 3A and B). Twin cortical grafts were placed between the spines of L3 and the sacrum, and one year later appear satisfactory (Fig. 4). However three years post-operatively (Fig. 5) a defect had developed in the neural arch of L3. X-rays five years post-operatively show this defect opening widely in flexion.

Recurrent Dislocation of the Elbow

By Geoffrey Platt, M.B., M.Ch.Orth., M.R.C.S., L.R.C.P.

Recurrent dislocation of the elbow is a rare condition. This is not surprising in view of the anatomical stability of the joint. Albert in 1881 described a case of recurrent dislocation of the elbow in which the radius and ulna displaced medially. In 1894 Heusner described one in which the radius displaced anteriorly and laterally, the ulna posteriorly and medially. In both these cases the displacement occurred in flexion. Bloch in 1900 was the first to describe an habitual dislocation in which both radius and ulna displaced posteriorly. Further cases were described from time to time and there are now approximately twenty of this type recorded in the literature.

In view of the rarity of the condition, a description of the following three cases may be of interest.

Case I.—Boy, aged 11 years.

Previous history.—Dislocation of the right elbow five times in five years, the first occasion being at the age of 6 years.

On each occasion the dislocation was reduced under anæsthesia and the elbow supported in a collar and cuff. On the third and fourth occasions it was immobilized in plaster for three weeks before applying the collar and cuff.

Each time the dislocation resulted from a fairly heavy fall.

Upon examination in June 1951 there was no deformity or muscle wasting of the arm. Flexion of the elbow was full and there was 10 degrees hyperextension present. There was 15 degrees lateral movement.

A transplantation of the insertion of the biceps to the coronoid process was performed, as described by Reichenheim, and the elbow was immobilized in plaster at 90 degrees for six weeks. It was then mobilized by active exercises. In ten weeks there was a full range of active movement including pronation and supination. There was still some lateral laxity.

It is now four and a half years since operation. There has been no further dislocation, despite numerous falls.

Case II.—Boy, aged 3 years.

The left elbow had become dislocated six or seven times, the first time being at the age of 9 months. It never resulted from a heavy fall but always from very minor trauma, once by his mother pushing up the sleeve of his cardigan. Reduction was always carried out with ease by the father.

Movements of the left elbow were full and the joint felt stable. It could not be dislocated with normal passive movements.

Transplantation of the insertion of the biceps was performed on 9.10.51 and the elbow immobilized in plaster for six weeks. Return of movements in this case was delayed by the formation of a keloid scar which required excision and grafting. It was twenty-seven weeks before full movements were obtained.

It is four years and two months since the operation upon this boy. During this time there has been no further dislocation.

Operative Findings in Case I and Case II

Case I.—General laxity of capsule and ligaments of the joint. Articular surfaces normal.

Case II.—Capsule and ligaments appeared to be normal but hyperextension of the elbow displaced the coronoid backwards, the olecranon acting as the fulcrum of a lever. Subluxation could easily be elicited.

In neither case was it possible to dislocate the elbow even when the joint was exposed. Only what was considered to be a "safe" degree of force was used.

Case III.—Girl, aged 16. I am indebted to Mr. Arthur Chance for the description of this case.

History.—In 1951 she fell from her bicycle injuring the left elbow. No details of her condition are known but she states that the arm was in plaster for fourteen days. She then received physiotherapy for three weeks.

From this time she complained of pain in the left elbow, limited extension, a creaking noise when bending and a weakness of the joint.

Examination.—The elbow was in normal position with normal movement except that there was hyperextension. On passive movement, both forearm bones could be subluxated backwards on the humerus. When the elbow is flexed it is in normal position but when it is extended it becomes subluxated, the ulna and radius slipping backwards and to the outer side.

On 30.11.54 operation was carried out. All the tissues down to the bone were found to be normal, except that the anterior capsule was tissue paper thin. The anterior lip of the coronoid was loose and was removed. A drill hole was made from front to back through the ulna and the biceps tendon threaded through this and caught by a button at the back. It would appear that the fracture of the tip of the coronoid process was the cause of the recurrent subluxation.

1.7.55: Seven months after the operation there is still some discomfort in the elbow and the patient cannot do more than light domestic duties.

In the extended elbow there is a considerable lateral rock suggesting a laxity of the ligaments but no antero-posterior displacement can be produced.

Ætiology

Sommer in 1928 classified the causal factors of the condition. He divided them into those cases in which there was no apparent bony change but in which laxity of the capsule permitted recurrent posterior displacement of the ulna on the humerus and those in which there was a definite bony abnormality.

The bony abnormalities were subdivided into

(1) Congenital malformation of the ulna (shallow trochlear notch).

(2) Bony changes due to trauma (fracture of external condyle or coronoid).

(3) Osteo-chondritis dissecans.

The most constant findings in the recorded cases are shallowness of the trochlear and ligamentous laxity (*see* Fig. 1).

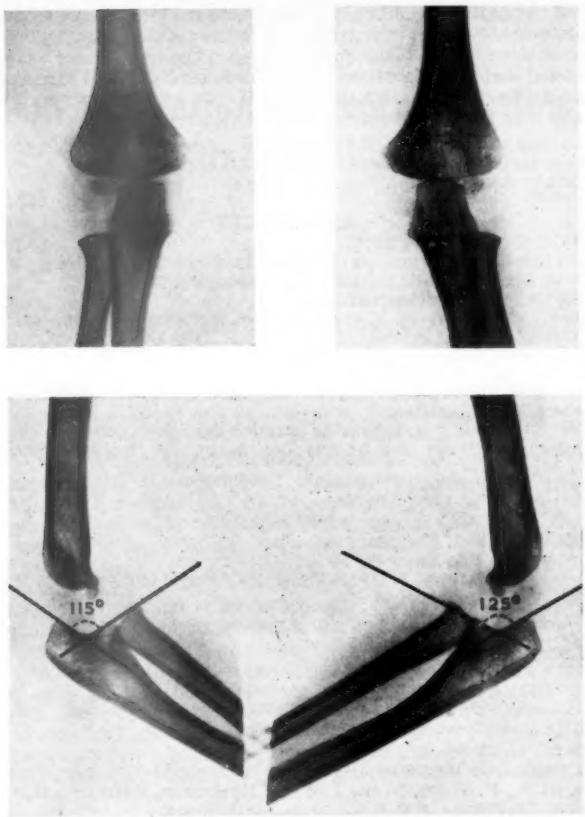


FIG. 1.—Lateral view of elbows of Case II. Upon superficial examination there appears to be little difference but upon measurement the trochlear notch is found to be flatter on the left side, the angle formed between the olecranon and coronoid processes being 125 degrees as compared with 115 degrees on the right side. The antero-posterior view of the elbows of Case II shows no abnormality.

It is suggested that recurrent dislocation of the elbow is due primarily to underdevelopment of, or damage to the trochlear notch or coronoid process. As with other joints, stretching of the capsule will occur with each dislocation and further decrease the stability of the joint. Lateral laxity due to lesions of the lateral ligaments or condyles of the humerus will increase the instability.

Treatment.—As with recurrent dislocation of other joints, the only satisfactory treatment is by operation.

Knoflach in 1935 was the first to operate upon a case of recurrent dislocation of the elbow. He plicated the lax capsule and further strengthened it by suturing strips of fascia lata across the anterior aspect of the joint.

Milch in 1936 treated a case by inserting a bone graft into the coronoid process, thereby lengthening it and deepening the trochlear notch.

Reichenheim in 1947 described his operation of transplanting the insertion of the biceps into the coronoid process. The strong pull of the biceps then prevents posterior dislocation of the ulna.

Kapel in 1951 described an operation in which he separated slips of the biceps and triceps which he passed through a hole drilled in the humerus and sutured to the olecranon and coronoid respectively.

In the 3 cases described, transplantation of the biceps was performed as it was thought

that plication of the capsule would be inadequate and the insertion of a bone graft into the coronoid would probably result in restriction of flexion of the elbow by bony block.

The Kapel technique had not been published when the first 2 cases were operated upon but it is considered that transplantation of the biceps tendon is still the treatment of choice as it is felt that strong muscle contraction drawing forward the upper end of the ulna must be more effective than fixed tendon clips which might stretch.

Finally, I would like to thank Mr. Arthur Chance for his kindness in providing me with the details of his case and for his permission to include them in this paper.

I would also like to thank Mr. J. C. Scott for his assistance and advice.

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These papers were fully discussed and among others who took part were the following: **J. I. P. James, M.S., P. Wiles, M.S., J. C. R. Hindenach, F.R.C.S., H. Osmond-Clarke, F.R.C.S., Miss E. H. Jebens, F.R.C.S., Professor J. Trueta.**

JOINT MEETING No. 2

Section of Pathology with Section of Orthopædics

Chairman—Sir REGINALD WATSON-JONES, M.Ch.Orth., F.R.C.S.
(President of the Section of Orthopædics)

[*February* 7, 1956]

DISCUSSION: GIANT CELL TUMOUR OF BONE AND RELATED CONDITIONS

Mr. Arthur L. Eyre-Brook (Bristol):

Some Clinical Impressions of Osteoclastoma, Reviewing a Small Series of Cases from the Bristol Bone Tumour Registry

Any discussion of osteoclastoma must include reference to the "brown tumours" found in hyperparathyroidism. Although resembling osteoclastomata closely in the radiograph, they are to be distinguished from these by frequently occurring well away from the ends of long bones, by the associated decalcification and thinning of the cortex and by the areas of fibrocystic change to be found in this and other bones. Confirmation comes with the blood chemistry investigations. We must always be prepared to exclude this diagnosis in any case of osteoclastoma.

Some confusion in the past has arisen as regards bone cysts, occurring in children and adolescents, most commonly in the upper ends of the femur and humerus, and found to have some osteoclastomatous material in the lining membrane. These cysts with excellent prognosis must not be confused with osteoclastomata, where the outlook is by no means so assured. The cysts form in the metaphysis, being displaced towards the centre of the shaft with the laying down of new metaphyseal bone in the ensuing years. The recognition of such a cyst is usually by fracture. The cyst is self-limiting and the only call for treatment is the need for a strong enough bone to withstand trauma in the future; evacuation of the cyst with the introduction of some bone chips usually provides a completely successful line of treatment.

Osteoclastomata in a long bone occupy that part of the bone formerly comprising the epiphysis; the patient is an adult and consequently no epiphysis exists, but it is the situation of the osteoclastoma extending up to the articular cartilage which presents the problem for surgical treatment. Whether the osteoclastoma starts in what was formerly epiphysis or metaphysis is debatable. Suffice it to say that these tumours in part occupy the area of the former epiphysis when presenting.

Cases of osteoclastoma, excluding those of jaw or skull, collected by the Bristol Bone Tumour Registry between 1946 *and* 1953 (Table I). There are 16 cases, confirming the comparative rarity of the condition. The sex incidence (10 females and 6 males) reiterates the usual preponderance of females. The average age is 31 years, with only 1 patient over 50 years and 3 below 20. 2 of these latter were situated in spinous processes of vertebræ, a situation where aneurysmal bone cysts tend to occur at this age; although one of these cases had large blood spaces in the microscopic section, both were accepted as osteoclastomata.

The tumours occurred in the usual sites in 10 cases:		The remaining 6 comprise:	
Lower end of femur ..	6	Spinous process and laminæ	2 children
Upper end of tibia.. ..	2	Upper end of femur ..	1
Upper end of humerus ..	1	Upper end of fibula ..	1
Lower end of radius ..	1	Lower end of humerus ..	1
		Sacrum	1

Reference will again be made to the case with an osteoclastoma at the lower end of the radius, as there was in addition an osteoclastoma at the lower end of the ulna and in the head of the 2nd metacarpal, both on the same side. Such an occurrence of multiple tumours in a girl of 17 years is just another astonishing feature in this surprising disease; observation over 7 years has given no support for any suggestion of hyperparathyroidism. One case, B.T.F. 254 (Figs. 1 and 2), showed the osteoclastoma crossing into a second bone, the lesion

TABLE I.—TABLE OF CASES

OSTEOCLASTOMA

Bristol B.T.R. 1944 to 1953. Excluding Jaw & Skull Lesions

BTR NO.	SEX & AGE	SITE	HISTORY	ONSET	BIOPSY	SURGICAL TREATMENT	RADIOTHERAPY	RESULT
62	M 48yrs.	Tibia. Ext. Tub	9 months Fracture.	1946	Open		5,600r. in 2 courses.	1955-Well. Carpenter
67	M. 34yrs.	Femur-Lower End.	3 years Fracture.	1944 1952	None Open	Amputation '52. Block Dissection Glands Fibro Sarcoma	3,680r. in 1 course	1946 to 1952 - Well Died July 1953
139	F. 18yrs.	Femur-Gt. Troch.	2 months.	1945	Open	Curettage '44	3,170r in 2 courses.	1955 - Well. Typist
154	M. 24yrs.	Humerus	4 months.	1948	Open		6,200r in 2 courses.	1955 - Well. Farm Worker
158	F. 17yrs.	Radius Lower End Ulna Metacarpal Head.	5 months.	1948	Open '53 Open '53 Open '48	Excision Ulna '53	8,800r. in 3 courses 7,500r. in 3 courses.	1953-Further Growth Ulna. 1955 - Well
190	M. 61yrs.	Femur-Ext. Condyle.	2 months.	1949	Open	After Fracture. 1950 Curettage & Bone Graft.	5,000r in 2 courses ('49-'50)	1955 - Well. Working.
219	M. 7yrs.	Spine - Lumbar Lamina	6 weeks.	1949	Open	Curettage '49	1,300 r. in 1 course	1955 - Well
241	M. 22yrs.	Fibula - Upper End.	1 year	1949	Open	Excision '49	None	1955 - Well
247	F. 34yrs.	Tibia - Upper End	9 months.	1949	Open	Curettage '49	3,000r. in 1 course.	1955 - Well
254	F. 42 yrs.	Sacrum	2 months.	1949	Needle		9,000r to 3 Fields in 3 courses.	1955 - Well.
394	F. 15 yrs.	Spine - Cervical Spinous Process	1 week.	1951	Open	Excision '51	None	1955 - Well.
404	F. 42 yrs.	Femur-Ext. Condyle	6 months.	1946	Open		4,900r in 3 courses.	Fracture 1948-Stiff Knee 1955 - Well.
423	F. 35yrs.	Femur-Lower End	4 months.	1951	Open	Curettage - Bone Chips - Nov '51. Amputation July '53.	5,850r in 1 course (6 Fields) 1952.	Lesion Extended 1952-53 Amputation 1953 1955 - Well.
630	F. 26 yrs.	Humerus - Internal Epicondyle.	10months.	1953	Open	Curettage & Cautery - May '53.	2,200r in 1 course Fracture 1953.	1956 - Well. Elbow lacks 60° Extension
659	F. 34yrs.	Femur-Int. Condyle	3 months.	1953	Open	Curettage - Bone Chips - Sept '53.	None	1955 - Well
671	F. 34 yrs.	Femur-Ext. Condyle	2 months.	1953	Open	Curettage & Cautery - Nov. 53.	1,260r in 1 course.	1955 - Well.

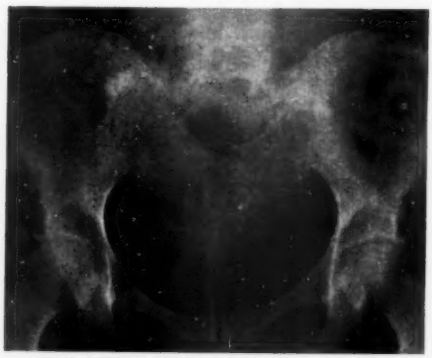


FIG. 1.—B.T.R. 254. Female. 42 years. Presenting with syndrome of pressure on cauda equina. Needle biopsy, January 10, 1951.

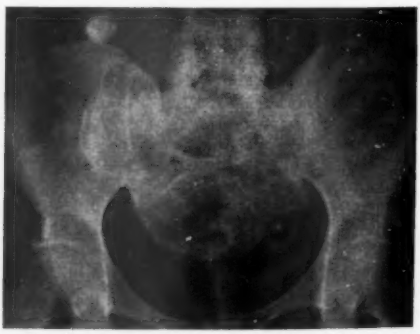


FIG. 2.—Same case, April 27, 1953, after radiotherapy (3,000 r in February 1950, June 1950 and July 1951—total 9,000 r. The spread of osteoclastoma into the right ilium is seen in both pictures.

occupying the whole of the sacrum spreading into the right ilium. In all probability, the spread occurred not across the joint but round the joint, as the articular cartilage always appears to present an effective barrier to the spread of an osteoclastoma.

The cases presented with pain or tumours, or a combination of these, and the average period of complaint was seven months. Two cases presented with pathological fracture. All our series save one were confirmed by biopsy; the histology has at times been difficult. Study of this clinical material did not support the idea that the osteoclastomata are a simple matter—far from it. Radiologists, pathologists and clinicians have all had second thoughts on reconsidering these cases; often what appeared malignant on radiological or histological grounds proved to be innocent on review. The radiological evidence of rapid growth and the disappearance of a limiting bony edge have been found to occur without implying malignancy, the one case of malignancy being only one of those showing a striking extension of the lesion within a few months.

Treatment:	Cases
Excision	2
Curettage and bone chips	1
Curettage, chips and radiotherapy ..	2
Curettage and radiotherapy	5
Radiotherapy	6

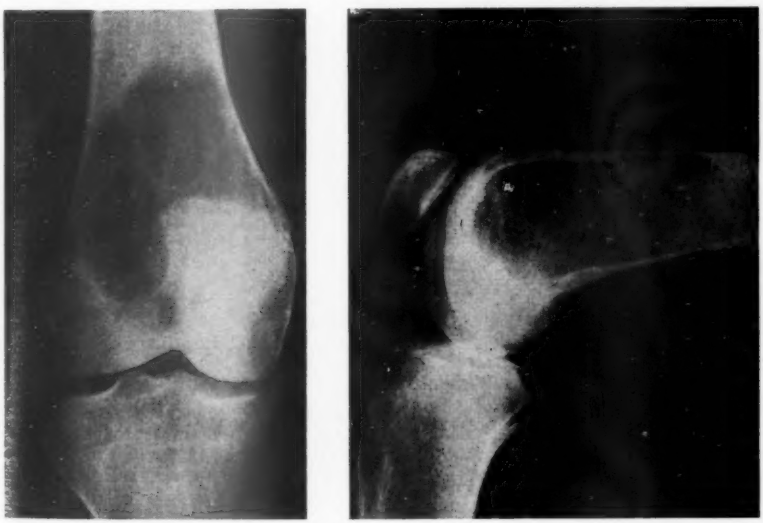
Those treated by surgery alone have not recurred, so far—two and a half years, four years and six years.

Of the 2 cases treated by curettage, chips and radiotherapy, one was being treated by radiotherapy when he sustained a fracture; curettage and bone chips were then employed in order to lessen the probable collapse. The second case proved to be malignant and amputation was subsequently performed.

The cases treated by curettage and radiotherapy varied from what was almost "operation cover" with radiotherapy amounting to 1,300 Röntgen units in 2 cases, to a dose of 3,000 Röntgen units in 2 others.

Those treated by radiotherapy alone received heavy doses as a rule and were treated by various radiotherapists. While not wishing to discuss the most suitable dosage, I wish to stress the high dosage in view of the subsequent biopsy in 2 cases. B.T.R. 423 was treated with 5,850 Röntgen units on the assumption of malignancy, substantiated by subsequent events.

Complications in this series comprise 2 cases of malignancy, local recurrence of the tumour in 1 case and 3 fractures. Reference must first be made to the case of malignant osteoclastoma. This case (B.T.R. 423) showed rather rapid growth when some previous radiographs were obtained for comparison with those taken when first in Bristol, and expansion of the bone was not present (Fig. 3). The histology left little doubt of the malignant nature of



A B

FIG. 3.—B.T.R. 423. Female. 35 years. Four months' complaints. No expansion of cortex. Radiographs six months previously showed tumour half the size. Malignant osteoclastoma.

FIG. 4.

FIG. 6.

FIG. 5.

FIG. 4.—B.T.R. 67. Male. 34 years. Case presenting with pathological fracture. May 8, 1944.

FIG. 5.—Same case, October 2, 1946, after radiotherapy (3,680 r in one course). Good sclerotic response to radiotherapy.

FIG. 6.—Same case, November 25, 1952, with fibrosarcoma developing at site of osteoclastoma. Some destructive change in bone, and large soft tissue tumours.

the tumour, but a thorough surgical curettage and the filling of the cavity with iliac chips had been performed by Mr. H. K. Lucas at the time of the biopsy. Fairly large doses of radiotherapy were used, but the radiograph showed a recurrence to be present and amputation was performed twenty months later. The patient shows no metastases after an interval of two and a half years.

A second case, B.T.R. 67 (Figs. 4, 5 and 6), the only case in which a biopsy was not performed, was diagnosed as a typical osteoclastoma on clinical and radiological grounds in 1944 and responded to radiotherapy (3,680 Röntgen units) with good sclerosis, this satisfactory picture persisting for over six years before the appearance of a rapidly-growing

FIG. 7.

FIG. 7.—B.T.R. 158. Female. 17 years. Osteoclastomata in lower end of radius and ulna. The lesion in the head of the second metacarpal is not seen in this radiograph, June 14, 1948.

FIG. 8.—Same case, showing sclerosis of all three lesions following radiotherapy. Radius and ulna: 8,800 r in three courses. Metacarpal: 7,500 r in three courses, December 2, 1950.

FIG. 9.—Same case, showing recurrence in ulna, August 13, 1953.

FIG. 8.

FIG. 9.

fibrosarcoma. This was confirmed by biopsy and treated by amputation, but death from secondaries occurred within a few months. It is difficult to avoid the association of the fibrosarcoma with the osteoclastoma, occurring in the same site and diagnosed eight years previously, but the relationship of the radiotherapy to this complication is debatable.

In 2, a biopsy was performed on an osteoclastoma following radiotherapy. In one case, B.T.R. 190, a fracture occurred thirteen months after 2,500 Röntgen units had been given and one month after a repeat dose of 2,500 Röntgen units, and this led me to evacuate the cavity, cauterize the walls and pack the cavity with iliac bone chips. The section obtained from the material removed showed very little change from the original biopsy.

In a second case, B.T.R. 158 (Figs. 7, 8 and 9), Mr. J. Bastow removed the lower end of the ulna when a recurrence occurred five years after what radiologically and clinically had appeared to be very successful radiotherapy. The doses were distinctly high, yet not only was there a recurrence in the ulna but when material was removed from the radius, osteoclastomatous tissue was found to be present here also.

What do these pictures of a sclerosed osteoclastoma really imply? This radius gave no radiological signs of recurrence but an inquisitive surgeon showed osteoclastomatous material to be present in quantity in the biopsy specimen. Yet this case was treated by radiotherapy in doses which many radiotherapists would consider excessive.

Fractures occurred in 5 cases, in 2 these comprised part of the presenting features of the case; in 2 cases, fractures complicated treatment by radiotherapy at periods of one year and two years after treatment was commenced. The fifth fracture complicated a case treated by curettage and radiotherapy.

We need to know more about the effects of radiotherapy on this tumour before we can decide as to its value. Well-planned surgery may give as thorough a clearance of the tumour with greater safeguards against fracture. Excision would appear to be the treatment of choice where it can be applied without loss of function, as in 2 of our patients. Excision with arthrodesis has a scope, but how often should we accept the loss of the joint? Finally, there are cases, as in the sacrum, where radiotherapy holds the undisputed field.

One cannot complete a review of this small series without again referring to case B.T.R. 158, where lesions occurred in the lower end of the radius and ulna and the head of the 2nd metacarpal on the same side. Biopsy of the 2nd metacarpal confirmed the diagnosis and the lesions were treated by radiotherapy. Recurrence five years later gave an opportunity to confirm the osteoclastoma in the other two bones. There was no suggestion of hyperparathyroidism then, nor in the subsequent seven years. What indeed is this tumour to present in three bones, grouped round one wrist?

The review of this small group certainly leaves one with doubts and anxieties over any case of osteoclastoma. Is it malignant? May it become malignant and what are likely to be the precipitating forces? Will a fracture complicate the picture and ruin the joint we seek to preserve? Again, what do these radiographs showing bony sclerosis after radiotherapy really imply? Are osteoclastomata in spinous processes and laminæ of vertebræ a group on their own to be found in children and adolescents?

In conclusion, I must offer my sincere thanks to my colleagues on the Bristol Bone Tumour Registry for their help in collecting and registering this series, to the many surgeons who have sent us cases to make up this small collection, and above all to Dr. C. H. G. Price, the secretary of the Bristol Bone Tumour Registry.

Dr. A. D. Thomson (Bland-Sutton Institute of Pathology, London):

The Pathology of Giant Cell Tumours

If the histological picture is regarded as the sole criterion for its diagnosis, the true giant cell tumour of bone is a comparatively rare lesion, and in the past thirty years there are only 34 proved examples in the records of the Middlesex Hospital (Thomson and Turner Warwick, 1955).

The excellent account by Jaffe, Lichtenstein and Portis (1940) established the specific histological pattern of these tumours and separated this pathology from the so-called "giant cell variants". Because of the frequency with which osteoclasts are encountered in diverse bone lesions, the true giant cell tumour has many histological imitators, but if the fibrous, the foamy, the cartilaginous, the ossifying and the other histological variants are excluded, a specific and potentially sinister tumour entity remains.

Age.—Giant cell tumours are most frequently encountered between the ages of 20 and 45 but they also occur in the older age group, when they are often associated with Paget's disease. These tumours are rare before the age of 20.

Sex.—There is an equal sex distribution, with 17 females and 17 males, in this series of patients.

Sites.—24 of the 34 tumours arose in the leg, and 20 of them involved the region of the knee. Four tumours originated in the humerus, and there were single examples of this tumour in the patella, a vertebra, the sacrum, the ilium, the scapula and the jaw. All the tumours in long bone arose from the distal or proximal ends and there was no example of a mid-shaft tumour.

Macroscopical appearances.—The end of the long bone is expanded by a hæmorrhagic tumour mass, which extends up to the articular cartilage although it rarely penetrates it. In the more aggressive examples the tumour commonly spreads through the cortex of the bone to invade the adjacent soft tissues.

Microscopical appearances.—The basic histological features of the giant cell tumour are the stromal cells, comprising oval and spindle-shaped cells among which there are giant cells of the osteoclast type.

Cellular variations in the stromal pattern are encountered which make it feasible to segregate these tumours into three histological grades. Grade I tumours show a regular

stromal pattern with no evidence of rapid or disorderly growth (Fig. 1). The Grade II tumours show a greater proportion of stroma, in which there is evidence of greater cellularity and some irregularity of the nuclei and a predominance of spindle cells (Fig. 2). The Grade III tumours show a frankly sarcomatous stroma, with atypical stromal activity, and the histological picture is that of an actively-growing tumour (Fig. 3).

The histological grading of these tumours gives some guide to their prognosis.

Of the 11 Grade I tumours, 5 recurred locally after treatment, but all responded to further therapy and there were no deaths due to this type of tumour.

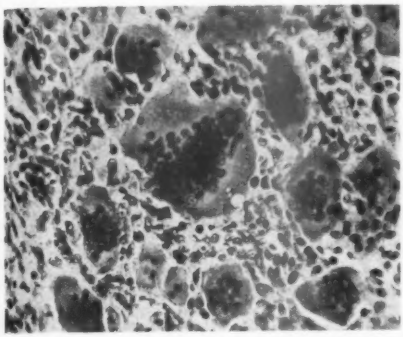


FIG. 1.—Giant cell tumour Grade I. Showing giant cells in a stroma of regular oval and spindle-shaped cells. × 300.

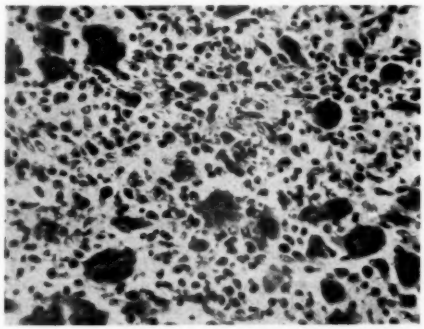


FIG. 2.—Giant cell tumour Grade II. Showing giant cells in a cellular stroma with evidence of nuclear irregularity and pleomorphism. × 300.

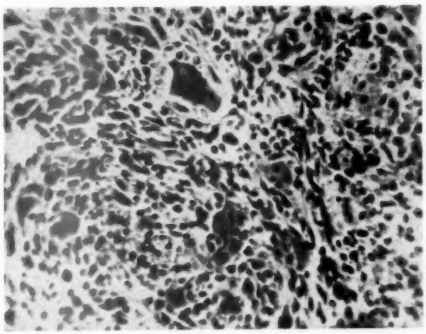


FIG. 3.—Giant cell tumour Grade III. Showing giant cells in a sarcomatous stroma. × 300.

There were 16 Grade II tumours, of which 11 recurred following treatment. 6 patients with this grade of tumour died, 4 because of metastases and 2 with local extension of the tumour in the pelvis and the chest respectively.

7 patients had Grade III tumours, and 4 of these died with metastases in spite of radical treatment of the primary growth.

Thus it would appear that the histological grade of the tumour does give some help in assessing the prognosis of these patients. However, it is by no means infallible, as can be seen in the Grade II type of tumour, 4 of which metastasized although their histological features were indistinguishable from their non-metastasizing fellows in the same grade.

Treatment.—The interest of this group of tumours is the high recurrence rate following local treatment. 11 patients were treated initially by curettage of the tumour and recurrence took place in 10 of these, while 15 patients had irradiation as the first therapeutic measure and 9 of these developed recurrence. One patient with a tumour at the upper end of the fibula had a resection of the upper part of this bone containing the tumour, but there was a recurrence later when the tibia was found to be invaded.

Prognosis.—Of the 34 patients in this series of giant cell tumours 8 died with metastases and 2 with fatal extension of the tumour in the pelvis and chest. 20 tumours recurred following treatment and 16 patients had amputations during the course of their treatment.

These prognostic details underline the sinister potentialities of these tumours and stress the importance of a definite histological diagnosis. If a representative portion of tumour tissue is submitted to the pathologist it should be possible to segregate the benign giant cell variants from the true giant cell tumours. Once the specific diagnosis has been made, an assessment of the stromal cytology of the tumour forms yet a further feature by which it is possible to gauge the prognosis and give a guide as to the advisability of more or less radical treatment depending upon the grade of malignancy of the particular tumour.

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A paper on "Aneurysmal Bone Cyst" was read by **Roland Barnes, F.R.C.S.Ed.**, Reader in Orthopædic Surgery, Glasgow University. This paper which includes several coloured illustrations can be consulted in the *Journal of Bone and Joint Surgery* (1956), **38B**, 301.

Dr. H. A. Sissons (Department of Pathology, Institute of Orthopædics, London):

Giant Cell Tumour Variants

The definition of giant cell tumour as a lesion characterized histologically by a spindle cell stroma and by frequent and evenly distributed multinucleated giant cells of osteoclast type, put forward by Jaffe, Lichtenstein and Portis in 1940, and already referred to in the present discussion by Dr. Thomson, leads to the exclusion of certain lesions which in the past have, rightly or wrongly, been included in this group. Among these excluded lesions are:

Certain cystic lesions
- Bone cyst.
- Aneurysmal bone cyst.

Certain fibrous lesions
- Solitary fibrous dysplasia with conspicuous giant cells.
- Non-osteogenic fibroma (the "xanthic" variant of giant cell tumour).
- Giant cell epulis.
- "Brown tumours" of hyperparathyroidism.

Certain cartilaginous lesions
- Benign chondroblastoma ("chondromatous giant cell tumour").
- Chondromyxoid fibroma (examples containing numerous giant cells have also been regarded as "chondromatous giant cell tumours").

Certain malignant bone lesions
- Tumours (particularly those encountered in Paget's disease of bone) possessing numerous tumour giant cells, but showing evidence of bony differentiation (i.e. unusual osteosarcomas).

In several of these instances, the confusion of such a lesion with giant cell tumour is clearly a matter of error. Thus a solitary lesion of fibrous dysplasia can sometimes masquerade as a giant cell tumour because of the presence in it of unusually frequent giant cells. I note the possibility of such an occurrence merely to draw attention to the care that must be taken in the histological evaluation of a lesion containing giant cells—particularly in a limited biopsy. In other instances, however, in particular with non-osteogenic fibroma and with benign chondroblastoma, we are concerned with the more fundamental problem of defining the relationship of the entities concerned—if entities they are—to giant cell tumour, and deciding in each case whether the separation of the particular lesion is justified. Do they, in fact, represent "variants" of giant cell tumour, or are they separate conditions which deserve to be known by special names?

Non-osteogenic fibroma of bone.—This name was given by Jaffe and Lichtenstein, in 1942, to lesions, usually occurring in the shaft of a long bone of a young patient, often appearing radiologically as a small eccentric lesion outlined by an encapsulating shell of bone, and characterized histologically by collagenous tissue arranged in whorled bundles and containing occasional multinucleated giant cells and collections of lipoid-containing phagocytes. The situation of the lesions, their occurrence in young patients, their uniformly benign behaviour, as well as their distinctive histological appearance, all contrast with giant cell tumour, and there is, in fact, little opportunity for confusion between the two conditions.

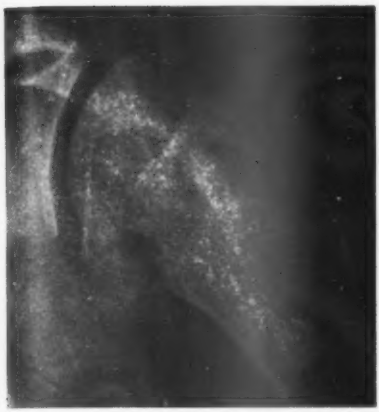

FIG. 1 (*Case I*).—X-ray of shoulder, showing osteolytic lesion of head of humerus.

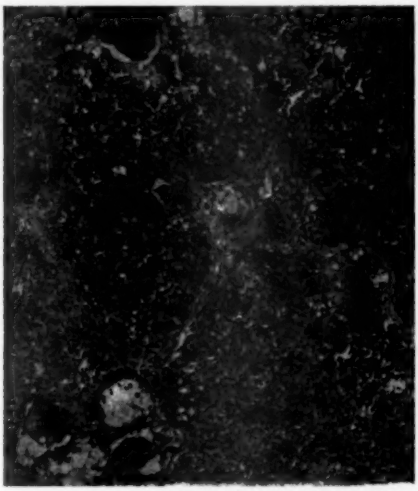

FIG. 2 (*Case I*).—Histological appearance of the tumour, showing predominantly round-celled tissue containing occasional multinucleated giant cells. (× 130.)

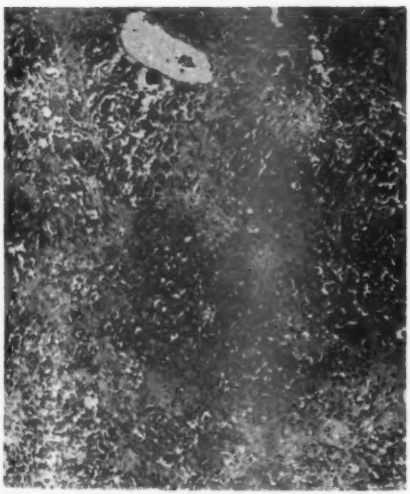

FIG. 3 (*Case I*).—An area of the tumour showing "chondroid" tissue with areas of focal calcification. (× 130.)

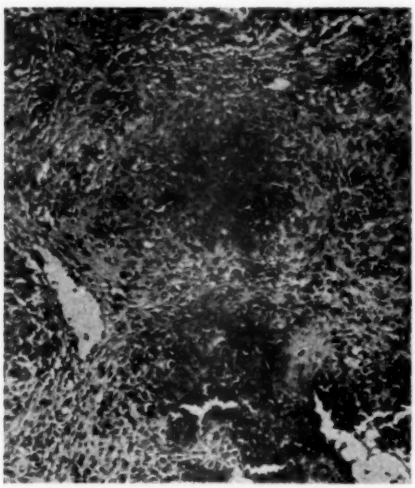

FIG. 4 (*Case I*).—One of the areas of focal calcification, showing the normal staining properties of cell nuclei. (× 150.)

Benign chondroblastoma of bone.—The lesion now referred to by this name is the one briefly noted by Ewing, in 1928, as the "calcifying giant cell tumour". The first detailed account of the lesion is that of Codman, who in 1931 described 9 cases under the title of "epiphyseal chondromatous giant cell tumour". Codman's cases all involved the upper part of the humerus, and, like Ewing, he regarded the condition as a "variant" of giant cell tumour. In 1942, Jaffe and Lichtenstein, from study of a further 9 cases, suggested that the lesion represented a specific tumour type separate from giant cell tumour, and designated it as "benign chondroblastoma of bone". They noted that the lesion consists of rounded cells which contrast with the spindle-celled stroma of the usual giant cell tumour, and that it

FIG. 5.

FIG. 5 (*Case II*).—X-ray of shoulder, showing osteolytic lesion of head of humerus.

FIG. 6 (*Case II*).—Histological appearance of the tumour, showing predominantly round-celled tissue containing occasional multinucleated giant cells. (× 150.)

FIG. 7 (*Case II*).—An area of the tumour showing rather fibrous "chondroid" tissue, and including an area of focal calcification with necrotic cells. (× 150.)

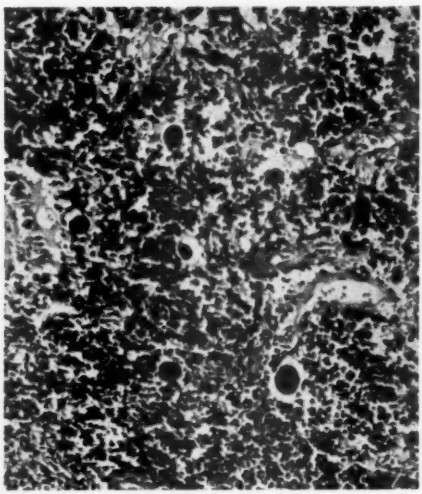

FIG. 6.

FIG. 7.

presents areas of chondroid tissue and focal calcification. Their observations led them to believe that the cells of the lesion were primitive cartilage cells—hence the name they applied to it. Although occasional giant cells were present in these lesions, Jaffe and Lichtenstein did not regard them as part of the "primary pattern". They showed that the lesion almost always involved the epiphysis of a long bone—not necessarily the humerus, however—that it occurred in patients below the age of 20 years, and that there was a remarkable preponderance of males in the observed cases. Radiologically, the lesions were osteolytic, and this, together with their situation at the ends of long bones, resulted in considerable similarity to giant cell tumour. The lesions reported were benign, local curettage being followed by cure, without recurrence or metastasis. It is important to note that the term "chondroblastoma" is used by Geschickter and Copeland (1949) to denote a quite different type of lesion—one for which other authorities would use the term "chondrosarcoma".

The following 3 cases appear to belong to the benign chondroblastoma group.

Case I (This is the case reported by Treasure (1955). I am indebted to Professor Gough, of Cardiff, for material and clinical information from it.)—Male, aged 14 years. History of pain in the left shoulder for one year. X-ray examination showed an osteolytic lesion of the medial aspect of the head and neck of the humerus (Fig. 1). The lesion was explored, and soft grey tissue, clearly

FIG. 8 (*Case III*).—X-ray of shoulder, showing osteolytic lesion of head of humerus.

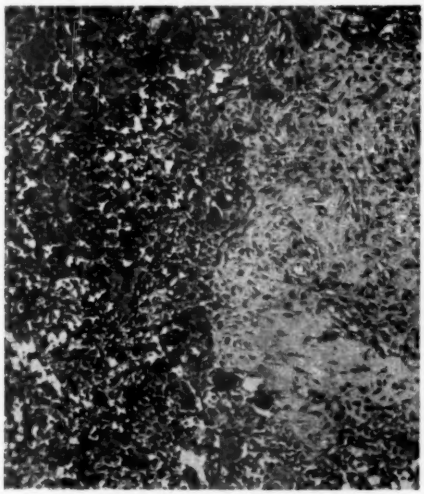

FIG. 9 (*Case III*).—Histological appearance of the tumour, showing cellular tissue containing giant cells. An area of rather fibrous "chondroid" tissue is present on the right side of the field. (× 150.)

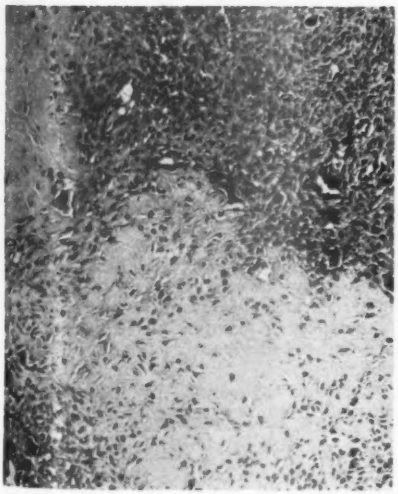

FIG. 10 (*Case III*).—A conspicuously fibrous area of tumour tissue. (× 150.)

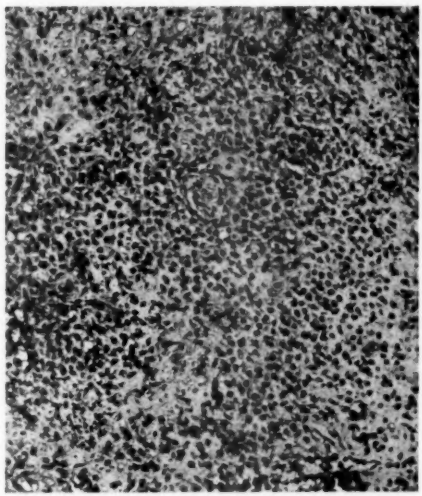

FIG. 11 (*Case III*).—Another area of the tumour, showing round-celled tissue with "chondroid" intercellular matrix. (× 150.)

demarcated from the surrounding bone, was removed by curettage. There was no recurrence two years after the operation. Histologically, the lesion is seen to consist of masses of small rounded cells whose appearance contrasts with the spindle-celled tissue of a usual giant cell tumour (Fig. 2). Occasional multinucleated giant cells are scattered throughout the tissue of the lesion. In some areas the tumour tissue has a "chondroid" appearance, the tumour cells being separated by connective tissue matrix showing a uniformly basophilic staining. In addition, scattered throughout the tumour there are areas of focal calcification (Figs. 3 and 4), and in some, but not all of these, tumour cells have lost their normal staining properties.

Case II.—Male, aged 14 years. History of pain in the left shoulder for nine months. X-ray examination showed an osteolytic lesion in the epiphysis of the head of the humerus (Fig. 5). The lesion was curetted, and a diagnosis of benign chondroblastoma was made on microscopic study of the tissue removed. Removal was incomplete, and the lesion had recurred two and a half years after operation. There has been no further recurrence three years after a second curettage of the lesion. Histologically, the predominant tissue of the lesion again consists of masses of small round cells with scattered multinucleated giant cells (Fig. 6). Areas of chondroid tissue are conspicuous, and patches of focal calcification, some of which contained necrotic tumour cells, are present (Fig. 7). The histological appearance of the tissue from the recurrent tumour was identical with that obtained initially.

Case III.—Female, aged 17 years. History of pain in left shoulder for six months. X-ray examination showed an osteolytic lesion involving the head of the humerus (Fig. 8). A diagnosis of benign chondroblastoma was made by means of biopsy, and the remaining tumour tissue was removed by thorough curettage. The patient is free from symptoms seven months after operation. The histological appearances are generally similar to those encountered in the two preceding cases (Figs. 9–11). The bulk of the tumour tissue consists of masses of rounded cells. Areas of focal calcification, though present, are infrequent in the present lesion, while areas of "chondroid" tissue are more conspicuous than in the previous cases. In some of these areas, the tissue, though showing a mild degree of basophilic staining, is rather fibrous, and the cells it contains are elongated or stellate in appearance (Figs. 9 and 10). In other areas, however, the "chondroid" tissue is less conspicuously fibrous, and its cells have the same rounded appearance as is evident in the more highly cellular areas of the tumour (Fig. 11).

It will then, I hope, be generally agreed that tumours of bone with this very distinctive histological structure do exist. They involve the epiphyseal regions of long bones in young individuals, and consist of round-celled tissue containing scattered multinucleated giant cells of osteoclast type, areas of "chondroid" tissue, and patches of focal calcification. They are frequently highly cellular lesions, and can consequently be mistaken for malignant lesions—in particular for malignant giant cell tumours—but their behaviour establishes that they are benign. Metastasis has not been reported in any of the published cases, and recurrence is rare despite the very local removal of tumour tissue that is usually carried out. In one case of a series reported by Hatcher and Campbell (1951), a malignant chondrosarcoma occurred at the site of a benign chondroblastoma three and a half years after irradiation, but the authors regard the malignant tumour as an independent post-irradiation sarcoma, and not as an example of malignant transformation of the benign tumour.

The cases I have presented are perhaps unusual—as were those of Codman—in that they all show involvement of the upper end of the humerus. I would like to emphasize that similar lesions do in fact occur in other bones, particularly the femur and tibia.

It is with the interpretation of the lesions that we are concerned. Our knowledge regarding their exact histogenesis is not complete, nor do we know the precise significance of the so-called "chondroblastic" tissue, the "chondroid" intercellular matrix, and the multinucleated giant cells which make up the tumour tissue. Such ignorance, however, is not infrequent in the study of connective-tissue tumours—particularly those of bone—and it need not stand in the way of practical identification and nomenclature of the conditions concerned. Histological subdivision is, in fact, carried as far as it is consistent with, or allows prediction of, other characteristics. Thus, while the separation of benign chondroblastoma from giant cell tumour depends ultimately on differences of histological structure, it is justified by the demonstration of differences of age and sex incidence, of distribution, and in particular of behaviour, between the two conditions.

The separation is of importance because of its prognostic implications, particularly as the benign chondroblastoma is sometimes, on histological grounds alone, confused with the malignant type of giant cell tumour.

In conclusion, not only must a clinical diagnosis of giant cell tumour always be regarded as needing histological confirmation, but the condition of benign chondroblastoma should be considered in the differential diagnosis when the lesion involves the end of a long bone in a patient under the age of 20 years.

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JAFFE, H. L., and LICHTENSTEIN, L. (1942) *Amer. J. Path.*, **18,** 205, 969.

——, ——, and PORTIS, R. B. (1940) *Arch. Path.*, **30,** 993.

TREASURE, E. R. (1955) *J. Bone Jt. Surg.*, **37B,** 462.

Dr. C. H. G. Price, in reply to the Chairman's remarks concerning the expediency of biopsy in the diagnosis and treatment of osteoclastoma, said that the problem relating to the propriety of taking a biopsy in such cases had been positively settled some years ago, and that the agreed point of importance for the patient was to establish, at the earliest moment, a firm diagnosis in any suspected bone tumour or quasi-neoplastic lesion.

The significance of the histological findings in a biopsy and their interpretation were a matter for further consideration. Their experience in Bristol had led them to the confirmed view that, to arrive at a correct diagnosis in skeletal tumours, it was essential to have complete agreement from all three lines of approach, surgical, radiological and histopathological, or that any serious differences between these opinions should be easily resolved by free discussion of the case with review of all relevant information. Furthermore, inability to reach such unanimity in the threefold diagnosis meant that the nature of the lesion was uncertain. This in itself was sufficient indication for complete reinvestigation. These conclusions are all the more cogent in relation to any destructive lesion in the major growing end of a juvenile appendicular long bone.

In the discussion of the value of histological grading of osteoclastomata which arose out of the paper read by Dr. A. D. Thomson, Dr. Price said that his experience in attempting the histological grading of bone tumours (mainly carried out, however, with osteogenic sarcoma) had indicated that it was possible to attain a fairly high level of correlation between the histological assessment of the degree of malignancy and the observed behaviour of a tumour when considered in terms of length of survival of the host.

It should not, however, be concluded from this that the histological grading was the only factor to bear in mind when estimating prognosis. It was essential also fully to appreciate certain other features, e.g. the type, precise site of origin, size, and stage of the tumour, the patient's age, the presence of concomitant skeletal disease, and possible response to treatment. There were doubtless other factors also, which were not at present known; but the grade of the tumour was only one of the components of the prognosis. This should depend upon consideration of both the tumour and its environment. Ability to assess the influence of certain of these other points had given him confidence in the practical value of the histological grading of bone tumours.

Mr. J. P. Jackson described a case of chondroblastoma of the hip, which clinically and radiologically had been confused with tuberculosis. Two biopsies were taken over a period of one year before diagnosis was established. The first gave no help as the wrong piece of tissue was taken. This exemplified the necessity for biopsy being repeated if necessary, and also that the pathologist could not help unless given suitable tissue from the tumour.

Mr. R. Weeden Butler made an appeal for biopsy of all bone tumours before treatment, not only because an exact diagnosis was in some cases obtained by this means, but because this would at least prevent the disastrous errors which occasionally occurred in atypical cases of bone infection which were given X-ray therapy as neoplasms. If biopsy were carried out, the pathologist should be given a fair chance of making a diagnosis by being presented with an adequate specimen of tissue, and he should not be expected to report usefully on minute fragments.

Mr. A. C. Bingold said that he had seen a giant cell tumour in the neck of the femur treated by local resection followed by the insertion of a massive endoprosthesis made of acrylic resin and steel. The fixation of this prosthesis to the femur was insecure, and it became loose after four years, so that a second prosthesis had to be inserted. Six years after the original operation the patient had no pain and there was no recurrence of the tumour. Although two of the speakers had shown that local resection was not always successful he said that he nevertheless felt that there was a case for treating giant cell tumours and other tumours of bone by resection of the tumour-bearing segment followed by insertion of an internal prosthesis.

Mr. T. Levitt said that there appeared to be a disagreement regarding the characteristics of giant cell tumours. Dr. Thomson had given them three grades of the tumour; while Mr. Barnes had asserted unequivocably that they were "agressive" from the onset.

It would be important to observe whether there were three distinct or discrete types, or whether they were progressive phases of the same condition. Did the study of recurrence in these tumours give any evidence of possible progression? Furthermore, how would such a concept of progressive phases of the same condition, if supported by adequate scientific evidence, affect prognosis and treatment?

Professor G. J. Cunningham asked Dr. Thomson whether he really believed the giant cells of osteoclastomata to be part of the tumour and whether he had found the histological structure of secondary deposits to be identical in giant cell content with that of the primary growth.

Mr. W. F. W. Southwood said that it had been shown that the behaviour of osteoclastomata could be predicted accurately by histological examination of biopsy specimens. If a tumour was found to be of Grade III malignancy, it would be almost certain to recur locally and to metastasize; it might be correct to treat these tumours by amputation.

Mr. T. J. Fairbank asked whether they would confirm the opinion that there was no place for a frozen section in the pathological diagnosis of a tumour in which the question of amputation had arisen.

Professor J. W. S. Blacklock said that the question whether one should have a biopsy or not must depend on whether a definite diagnosis could be made from the clinical and radiological evidence. If this were not possible, a biopsy should be taken: an adequate amount of tissue should be sent to the pathologist, as tumours in bone might vary a great deal in structure in different areas. Owing to the very cellular nature of many of these tumours, frozen sections were not very satisfactory, as the tumour broke up into small pieces, and under such circumstances a pathological diagnosis might be indefinite and recourse must be made to paraffin sections. In the diagnosis of these tumours it was essential that the clinical, radiological and pathological evidence be considered together: failure to do so might result in a limb being amputated unnecessarily.

Mr. F. G. St. Clair Strange asked if, as Mr. Barnes had suggested, aneurysmal bone cyst were the result of hæmorrhage into bone, there might not be similar appearances to corroborate this theory in the pseudotumours of hæmophilia, which were due to hæmorrhage into bone. He also recalled the difference of opinion of the leading articles in the *Journal of Bone and Joint Surgery* of 1949 as to the site of origin of the tumour, whether epiphyseal or metaphyseal, but interpreted the feeling of the meeting as evading the question successfully by placing the onset at above 18 years of age, when most epiphyses had fused. He also asked whether the employment of radiotherapy, after an osteoclastoma had been scraped out and packed with bone chips, was a rational procedure.

Mr. Norman Capener stated that it should not be thought that there was a hard and fast rule that giant cell tumour was not found in children under 16, or that it was never metaphyseal in origin. He had recently come across such a tumour occurring in the lower metaphysis of the tibia of a child aged 8. After a most thorough removal, and packing with iliac bone, the tumour had recurred with clear aggressive characteristics.

Dr. Thomson, in reply to Mr. Levitt, said that he regarded all giant cell tumours as either aggressive or potentially aggressive. In reply to Professor Cunningham, he said that the precise nature of the giant cells in a giant cell tumour was unknown, but they were usually regarded as either osteoclasts or as tumour giant cells formed by aggregation of the stromal cells. Whatever their origin, these giant cells appeared to form an integral part of the tumour process as they were always represented in the tumour tissue, whether this be primary, recurrent or even metastatic.

Section of Dermatology

President—Louis Forman, M.D., F.R.C.P.

[*December* 15, 1955]

Dermatomyositis with Malignant Disease.—O. L. S. Scott, M.B., M.R C.P.

Man, aged 73.

Complained of infiltrated, erythematous, œdematous rash on the face, scalp and "V" of neck for the past five months. He has no rash elsewhere. For three months he has noticed gradually increasing myasthenia. He has great difficulty in raising the arms, and there is muscle wasting which is obvious in the hands. For several years he has had a cough.

Investigations.—(1) X-ray of chest shows a shadow in the left upper segment ? neoplasm. (2) Biopsy of forearm shows degeneration of muscle fibres. (3) He has a well-marked creatinuria and raised blood creatine level. The urinary creatine, carried out on five successive days, averaged 380 mg. per day. Test administration of creatine showed faulty, incomplete storage (Dr. J. N. Cumings).

This patient has dermatomyositis. However, a biopsy of the unusual skin eruption is needed since it does resemble a "reticulosis". The question of lung tumour will have to be revealed by bronchoscopy and/or the passage of time. If confirmed, it will be interesting to learn whether X-ray treatment of the tumour will influence the myositis as in the 4 cases quoted by Dr. Dowling in his Watson Smith Lecture (1955). In his own series of 20 patients with dermatomyositis at St. Thomas's Hospital there were 3 with cancer. In these, as in the other 27 which he quotes, death occurred from the dermatomyositis and not from the cancer. Furthermore, the cancer seemed always to commence before the onset of dermatomyositis.

Postscript (Dr. I. W. Whimster).—Biopsy of skin from the neck was consistent with dermatomyositis.

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Dowling, G. B. (1955) *Brit. J. Derm.*, **67**, 275.

Dr. F. F. Hellier: I had a case which is similar to this though clinically more typical of dermatomyositis; this case presented by Dr. Scott looks more like leukæmic infiltration of the skin of the face. My patient had a cancer in the abdomen and dermatomyositis but before she succumbed the dermatomyositis cleared up.

It is very interesting that tumours have these effects. Sometimes one sees pruritus with tumours, sometimes a pemphigoid eruption or some other skin manifestation; the attention of our physician colleagues might be drawn to these cases. If a tumour can influence the skin in this way it may affect other organs; thus we might have a tumour inducing hepatitis and all sorts of other possibilities come to mind.

I would suggest doing an electromyogram to see whether the changes are those of a myopathy or a neuropathy. The latter is found associated with bronchial carcinoma but the former is true dermatomyositis.

Hydrargyria.—J. A. Cardno, M.D., and R. H. Meara, M.R.C.P.

Miss A., saleswoman, aged 46.

History.—Nine years ago the patient noticed that the margins of the eyelids were becoming discoloured. This discoloration now involves most of the eyelids. She revealed that she had been applying "Golden Eye Ointment" to the margins of the eyelids every night since she was 7 years old.

On examination.—The upper and lower eyelids on both sides show a slaty pigmentation, most marked just beyond the lid margins.

Investigations.—A biopsy was performed from the edge of the pigmented area of the right upper lid.

Section 1 shows pigment granules throughout the corium mainly in cells which show long dendritic processes. There is some concentration of pigment just below the epidermis and around hair follicles.

Section 2 has been treated with iodine and sodium thiosulphate and shows that the pigment has been completely removed by this treatment.

Comment.—Pigmentation from the topical application of creams containing calomel was reported by Goeckerman in 1922 and in 1925. In the later report he describes histological changes similar to those in the present patient and showed that the pigment was removed by

compound solution of iodine. This does not distinguish the pigment from silver but the history in the present case indicates that it must be mercury.

We are indebted to Dr. G. C. Wells for suggesting and performing the histological investigations.

REFERENCE

GOECKERMAN, W. H. (1922) *J. Amer. med. Ass.*, **79**, 605; (1925) **84**, 506.

Dr. G. C. Wells: The pigment is in the form of fine granules widely scattered throughout the corium. In many places beaded strands of pigment are seen and some of these strands can be related to histiocytes and the arrangement suggests that much of the pigment is contained within phagocytic cells with long trailing processes (Fig. 1). The pigment granules show up brilliantly

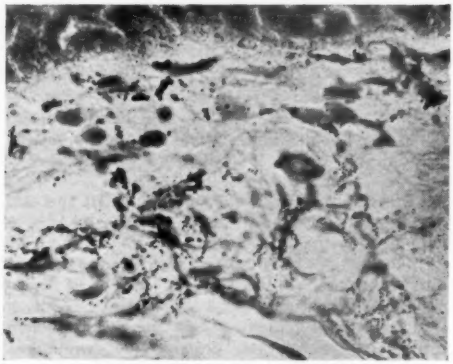

FIG. 1.—Hydrargyria. ×1,400.

under the dark field microscope. Treatment of the section with iodine completely removed all pigment and this suggests that it is metallic but it would not distinguish between silver and mercury. If the metallic pigment is mostly within the histiocytes it seems to me that there is a likelihood of gradual resolution once the applications have been stopped. The metal is in some form which is chemically rather inert and it does not give the colour reactions for mercury. It may be present as a proteinate or perhaps as an oxide.

Primary Tuberculosis of the Skin.—R. H. MARTEN, M.R.C.P. (for D. I. WILLIAMS, F.R.C.P.).

C. H., girl aged 12 years.

History.—Seven weeks ago she developed a small spot on the left lower face which was described as being like a boil and discharged some "pus". Five weeks ago a painless lump which has since become larger was noticed under the chin. She was given Terramycin for one week without improvement. She has no cough, is gaining weight and her general health is satisfactory.

Past history.—Three quinsies two years ago.

Family history.—Earlier this year her mother had "pneumonia" but is now apparently quite well. No known tuberculosis in the family.

On examination.—In the region of the left lower jaw there is a small oval lesion measuring 5 mm. in diameter. The centre is pale and slightly depressed and the edge is brownish yellow but not raised above the surrounding skin. There is no induration. The left submental glands are enlarged, fluctuant and matted together and have become attached to the overlying skin which is slightly pink. There are no other abnormalities apart from a very occasional spike of pyrexia.

Investigations.—Blood count: Hb 108%, W.B.C. 5,600, normal differential count. E.S.R. —10 mm. in one hour (Westergren). Mantoux test—1 : 10,000 negative, 1 : 1,000 positive. X-rays of chest and neck revealed no abnormality.

Treatment.—Isoniazid 50 mg. t.d.s. and streptomycin 0·5 gram intramuscularly daily for the past ten days have produced some slight decrease in the size of the glands.

Comment.—There still seems to be a difference of opinion as to whether uncomplicated primary tubercle in the lung should be treated or not, but in the present case we felt that isoniazid and streptomycin might cause resolution of these glands even at this stage, although it seems likely that surgical intervention will be required later.

POSTSCRIPT (April 26, 1956).—Treatment with streptomycin, isoniazid and para-

amino-salicylic acid is being maintained. The skin lesion is cleanly scarred with a slight peripheral staining. The affected glands have shrunk to a very firm, pea-sized nodule, difficult to locate.

Dr. Brian Russell: When the lesion is examined with a diascope, apple-jelly nodulation is visible. I think the condition is changing from a primary tuberculous chancre to lupus vulgaris. On May 15, 1952, I showed a boy to this Section with a primary tuberculous lesion which was undergoing similar changes, coinciding with the conversion of his Mantoux reaction from negative to positive. I think this patient should be treated with isoniazid for one year at least.

Dr. D. I. Williams: I agree with Dr. Brian Russell: there is certainly already the softening and the colour of lupus vulgaris.

Dr. B. C. Tate: If the glands show signs of settling it is better not to treat them so that the child develops full immunity. If the local condition is deteriorating and the glands are increasing in size one should treat.

Blue Nævus.—W. G. TILLMAN, M.D.

I. McC., female, aged 20.

This patient first attended a surgical clinic on January 25, 1955, for a painful tender lesion on the back. She stated that the extensive nævoid abnormality on her back had been present ever since birth.

In view of the painfulness of one of the large easily palpable nodules she was admitted to hospital on September 1, 1955. The nodule was excised and histologically examined. A pathological diagnosis of neurofibroma was made. A section was then submitted to Dr. H. Haber who reports: Blue nævus, cellular type.

Comment.—If I had made the diagnosis of blue nævus originally I should have advised the surgeon even more strongly not to perform an operation on this lesion. Some years ago I had arranged to show a case of blue nævus to the Section but the patient fell ill on the day of the meeting. He died of malignant melanoma some months later. The original biopsy had not shown anything apart from the histology of blue nævus.

Cases of blue nævus terminating as malignant melanoma have been reported in the past, but they must be rather rare. In this case the deep blue colour should have suggested the diagnosis to me, even if I were not familiar with the very large nodular and more superficial lesions which this case shows.

Dr. H. Haber: The lesion shows the following features:

There is a central core of round and spindle-shaped cells, resembling nævus cells in a dermal cellular nævus and fibromatous tissue respectively. In addition to this one can see masses of melanin-containing spindle-shaped cells. The lesion is, therefore, a blue nævus cellular type. One might perhaps also consider a pigmented neurofibroma.

Pigment cells are found in both epidermal and dermal layers and also, particularly in lower vertebrates, in perineural and perivascular layers as well as in the cœlomic wall. This lesion represents a malformation and is perhaps an atavistic relic consisting histologically of neurofibromatous tissue and melanoblasts.

The so-called nerve nævus of Masson is a cellular nævus showing typical nævus cells as seen in a common mole, but in addition to this one can observe *lames follacées* and neuroid tubes, a neurofibromatous tissue within which some of the aforementioned cells are embedded. According to Masson the lesion represents an attempt to form nerve ends. The common mole and the nerve nævus may be pigmented, the pigment obviously deriving its origin from dermal melanoblasts which in this case are present in such lesions.

So there is a certain parallel to be drawn between a pigmented common mole or nerve nævus and a blue nævus cellular type. Embryologically all the cells mentioned derive their origin from the neural crest.

The general pathologist could, therefore, call this lesion a pigmented neurofibroma; the dermatologist a cellular blue nævus. Substantially it means the same.

The following cases were also shown:

Poikilodermia Congenitale.—Dr. ARTHUR ROOK and Dr. R. A. DAVIS.

Acquired Epidermolysis Bullosa.—Dr. J. H. ALLISON (for Dr. F. RAY BETTLEY).

Pre-mycotic Eruption.—Dr. P. F. BORRIE.

Senear-Usher Syndrome.—Dr. E. N. M. JOHNSTON (for Dr. E. W. PROSSER THOMAS).

Epidermodysplasia Verruciformis Together with Dysplasia of the Lymphatic System.—Dr. I. S. HODGSON-JONES.

Orf (or **? Milker's Node**).—Dr. BRIAN RUSSELL.

(1) **Necrobiosis Lipoidica.** (2) **Dermatitis of the Scalp and Face Following the Use of Selsun.**—Dr. LOUIS FORMAN.

A short paper on **Methods of Measuring Blood Flow** was read by Dr. H. E. HOLLING.

[*January* 19, 1956]

Erosive or Desquamative Gingivitis. Lichen Planus of the Scalp and Body.—LOUIS FORMAN, M.D., F.R.C.P.

Mr. G. B., aged 43. Eighteen months ago there was a generalized lichenoid eruption of the body and scalp. This was followed by considerable hair loss and an acute pustular dermatitis of the scalp which appeared to be provoked by an actinic reaction. The changes on the gum of the upper jaw have been noted for three years.

FIG. 1.—Atrophy of the convexity of gingival mucous membrane.

On examination.—A few lichen planus papules are to be seen on the genitals and on the chest. Pigmented macules on the body represent faded lichen planus papules. The scalp shows numerous atrophic areas leading to considerable thinning of the hair. The convexity of the gingival mucous membrane of the upper jaw is atrophied with a leukoplakic border which is not, however, very well accentuated (Fig. 1).

Comment. — Erosive gingivitis has been recognized as a localized manifestation of lichen planus in the mouth. There is a fixed, thinned, reddened and even ulcerated epithelium, occupying the convexity of the alveolus. The actual gum margins may be spared.

There may be a leukoplakic border showing the characteristic pattern of lichen planus. It may be associated with a similar process on the mucous membrane of the cheeks. This was well seen in the severe case shown in 1952 (Forman, 1955). This patient had loose teeth and a defined atrophy affecting the gum margins around these teeth. On the left cheek was a patterned leukoplakia. On the right cheek, there was an extensive superficial ulceration with a thick leukoplakic rim, exhibiting the extensions of lichen planus. The labia minora showed similar changes.

Biopsy of a line of barely visible papules on the arm gave the histology of resolving lichen planus.

The interest in this patient is the involvement of the alveolar epithelium for some eighteen months before the acute dissemination of lichen planus occurred.

REFERENCE.—FORMAN, L. (1955) In: Proceedings of the 10th International Congress of Dermatology, 1952. London; p. 468, Case No. 67.

Dr. I. J. Merenlender: This patient was under my care for more than a year. When first seen in October 1954 he had numerous typical lesions of lichen plano-pilaris et follicularis over the trunk, upper extremities and penis. In the mouth he showed the same gingival lesions as seen on this occasion. On the scalp there were several circumscribed erythematous scaly patches with secondary pyodermia and other small atrophic bald areas, which reminded one of pseudopelade (Brocq). No lichen planus elements were seen on the scalp. His urine and blood count showed no abnormality and culture from the pyodermic lesions revealed a coagulase-positive *Staphylococcus pyogenes*. The pyodermic lesions responded to a course of penicillin but this had no effect on the lichen planus, which cleared in four months from trunk and extremities after courses of Acetylarsan and Mapharside; Kromayer lamp treatment was given locally. No mercury was given. This treatment had no effect whatever on the lesions in the mouth and on the scalp.

Dr. R. M. B. MacKenna: May I ask whether you have any idea how many cases of the exanthematous types of lichen planus which have been given mercury tend to develop gingivitis?

The President: This patient noted the soreness of the gums before treatment was given.

Dr. G. B. Mitchell-Heggs: It seems to me that the atrophy round the gums has nothing to do with the treatment of lichen planus with mercury. If it is postulated that mercury is the cause, I should say that for quite a number of years Sir Ernest Graham-Little used Enesol, and at that time many people did; I do not remember seeing any serious consequences such as atrophy of the gums in patients treated with this drug. I have also seen these gum changes in patients who have not had any arsenic or mercury therapy.

Dr. I. B. Sneddon: We have been doing a study on treatment in lichen planus and the results may be published by Dr. Ronald Church.

So far, some 120 cases of lichen planus have been treated, some with only a placebo. In about one-third of the cases, recovery occurs speedily whatever form of treatment is used and this is borne out by other published series. A certain number become chronic whatever treatment, including cortisone, is used.

Dr. P. D. Samman: I have been doing the same thing and I agree with Dr. Sneddon.

Sarcoidosis Presenting as Scar Granuloma.—ARTHUR ROOK, M.D., and R. A. DAVIS, M.B., B.S.

S. S., male, aged 69. Retired general labourer.

History.—In 1917 the patient suffered a powder-burn of the right thigh and a heavily-contaminated shrapnel wound of the left forearm, which was long in healing. Thereafter his health remained excellent. In September 1955 a red irritable patch appeared on the lower lip and red nodules developed at the site of the old wound of the left forearm and small papules appeared above the right knee. All the lesions increased in size but caused no discomfort. In October a red irritable patch developed on the right upper lip and a nodule on the nose. There has been no cough or shortness of breath.

On examination (1.11.55).—Skin: A well-defined dull red indurated plaque with superficial crusting on the right upper lip extends to the cheek and to the vermilion border. Outlying nodules are visible and palpable. There is a small nodule to the right of the tip of the nose. The lower lip is slightly thickened and an irregularly nodular plaque extends from the vermilion border to the right side of the chin. There are a number of reddish-brown slightly scaly papules on the front of the right thigh, just above the knee. On the flexor surface and ulnar border of the left forearm are three linear scars in and around which are numerous papules and nodules. Some of the latter are superficial and others are entirely subcutaneous in situation. The largest are 2 cm. in diameter. A solitary subcutaneous nodule is palpable on the flexor aspect of the right forearm. Extension of all fingers of the left hand and flexion of the ring and little fingers is limited.

No abnormality was discovered on general examination. There was no palpable enlargement of the spleen or superficial lymphatic glands. Ophthalmic examination by Mr. G. F. Wright showed no evidence of sarcoidosis.

Investigations.—X-ray chest (Dr. J. M. Allcock): "There is rather doubtful hilar glandular enlargement on the left side and some linear streaking is spreading out from the left hilum to the lung field. The appearances are consistent with a sarcoid. Hands and feet: No sarcoid lesions seen in the bones. Left forearm: Three radiopaque foreign bodies are present lying posteriorly in the middle of the forearm. There is considerable vascular calcification."

W.R. and Kahn negative. Red and white blood counts within normal limits. E.S.R. 29 mm. in one hour (Westergren). Serum proteins 8·1 grams/100 ml. Normal electrophoretic pattern. Mantoux: 1 : 1,000 negative, 1 : 100 positive.

Histology.—Nodule, left forearm (Dr. R. A. Parker): "A piece of skin, most of the corium of which is replaced by sharply demarcated foci of epithelioid and giant cells, some of the latter containing small oval clear spaces in their cytoplasm. A few foci show early necrosis. Occasional small particles of doubly refractile material are present in the foci. I consider these changes compatible with the clinical diagnosis of sarcoidosis."

Comment.—Löfgren *et al.* (1955) found cutaneous and subcutaneous scar granulomata in 17 of 400 patients with sarcoidosis and they found evidence of pulmonary sarcoidosis in all of 6 patients presenting with the complaint of swelling in old operation scars. Particles of quartz or talc were discovered in the biopsies from many but not all the scar granulomata and the origin of some of the scars make it unlikely that they had been contaminated by mineral particles; it seemed probable that trauma alone could determine the localization of sarcoid lesions. Our patient gave no history of any injury to the face. He would appear to have systemic sarcoidosis, the localization of some but not all lesions being determined by previous trauma with or without the presence of silica compounds in the tissues.

REFERENCE.—LÖFGREN, S., SNELLMAN, B., and NORDENSTAM, H. (1955) *Acta chir. scand.*, **108**, 405.

Dr. R. D. Sweet: This case is one of the few in which there are sarcoid lesions in places other than where trauma occurred. I have always felt with these cases that it is not really a reaction to silica slowly dissolved over many years, but that the patient is going to get sarcoidosis anyway, or is a borderline case, and the presence of the foreign bodies merely determines the localization. I have seen quite a number of them; I do not think they are rare. Several have cleared up spontaneously. In one case lesions were localized to the lower lip and upper lip. The lesion in the lower lip was excised and the upper lip was left alone in order to see what would happen (it was part of the site of the original injury), and the lesion faded away after about eighteen months. The patient had no other lesions anywhere.

In a case I showed in 1950 a strong point was made of the fact that the sarcoid nodules in the section did not seem to be centred around the foreign bodies there. It was not felt to be a matter of trying to gobble them up and get rid of them. They were lying there higgledy-piggledy, quite irrespective of the reaction going on.

REFERENCE

SWEET, R. D. (1950) *Proc. R. Soc. Med.*, **43**, 173.

The President: You do not think the foreign bodies had any part in the granulomatous reaction?

Dr. Sweet: Not directly, only by creating a site of lowered resistance.

Dr. T. Kindler: I showed a case in this Section in 1955 of an exanthematous sarcoid, which developed after sun-exposure, strictly in the exposed areas. I suggested as a possible explanation of this localization that in this patient, who was already suffering from sarcoidosis of the lungs, the trauma of a severe sunburn was acting as a predisposing factor for the outbreak of the cutaneous eruption.

REFERENCE

KINDLER, T. (1955) *Brit. J. Derm.*, **67**, 149.

Lupus Miliaris Disseminatus Faciei.—C. M. RIDLEY, B.M., M.R.C.P. (for BRIAN RUSSELL, M.D.).

F. H., housewife, aged 46. Since 1950 she has had a persistent eruption on the face, which at first was papular but later became maculo-papular. The lesions have never ulcerated.

General Health.—Good. She suffers from migraine and has had urticaria of unknown causation since 1953. In 1944 she had a papilloma removed from the right vocal cord. She has 3 children, all well, and has had one miscarriage.

Family history.—A step-sister died twenty-five years ago from pulmonary tuberculosis. A sister has pulmonary tuberculosis.

Investigations.—Biopsy: Epithelioid tubercles in the upper dermis with a marginal zone of lymphocytes. Wassermann reaction negative. X-rays of chest and hands normal. Blood count normal. E.S.R. 10 mm. in one hour (Westergren). Plasma protein: 6·5 grams/100 ml. Mantoux test negative at 1 in 10,000: positive at 1 in 1,000.

Past treatment.—In 1950 she had a course of calciferol 100,000 units daily and in 1953 a course of isoniazid, 300 mg. daily, for over six months. There was no response to either treatment. During 1950 and 1951 some lesions were treated with the cautery.

On examination.—Somewhat hirsute woman with guttate, reddish-brown macules and papules, showing a brown colour on diascopy. The lesions are irregularly scattered on the cheeks, the chin, the eyelids and the bridge of the nose. There are a few comedones and small scars.

Comment.—The lesions did not respond to calciferol or to isoniazid. This is of interest in view of the success of both drugs in lupus vulgaris and that recently reported of isoniazid in erythema induratum, conditions in which a tuberculous ætiology seems more firmly established than in lupus miliaris disseminatus faciei.

Dr. G. B. Dowling: I believe that so far no one has succeeded in proving the tuberculous causation of this condition. More often than not the existence of tuberculosis elsewhere cannot be demonstrated and the condition is uninfluenced by antituberculosis remedies; even the reaction to tuberculin may be negative.

The President: Small papular crusted lesions, sometimes disseminated over the face and ears, may appear in children with miliary tuberculosis during treatment with streptomycin.

Multiple Kerato-acanthomas.—P. A. J. SMITH, M.B., M.R.C.P.

S. A., male, aged 36. Furniture remover.

For over fourteen years he has had recurrent papules and nodules on the face, scalp, and limbs. They begin as blackheads or follicular horny plugs with an erythematous halo. At first caseous matter may be expressed, but after a few weeks they become hard and dome-shaped with the keratotic centre of molluscum sebaceum. The trunk has been free, but the lesions have not been confined to the light-exposed parts of the limbs. During six years of observation at the London Hospital no lymph nodes have been enlarged.

When serving in Egypt in 1942 the condition was diagnosed as molluscum contagiosum and as new lesions appeared over several years they were treated with phenol. Subsequently excision, cauterization, curettage and radiotherapy have been used. None has been seen to disappear spontaneously but several treated by methods other than excision have recurred.

He has never worked in contact with tar or pitch, and there is no relevant family history.

On examination.—Right forearm: dome-shaped pink papule 3 mm. in diameter. Left forearm: keratotic follicular papule with an erythematous halo. Forearms, legs, thighs, neck and face: scars of treated lesions.

Histology.—In six years twenty-five lesions have been examined. Clinical diagnoses included giant molluscum, button epithelioma, sebaceous cyst, and kerato-acanthoma. Pathologists have reported squamous papilloma, horny cysts in squamous epidermal downgrowths or low-grade squamous carcinoma. Dermal infiltrate, when present, is slight.

Comment.—This disease seems to belong to a group of conditions characterized by the presence of one or more tumours with a definite clinical and histological appearance. The early lesion is a dome-shaped papule with a central keratotic crater. The microscopical

picture may be that of a benign squamous papilloma or downgrowths into the dermis may arouse suspicions of malignancy. Nevertheless only extremely rarely does the lesion become a true squamous carcinoma with metastases to the regional lymph nodes.

Four types of case may be recognized in this group:

(1) Single molluscum sebaceum (MacCormac and Scarff, 1936) on exposed parts.

(2) Tar molluscum, which may be single or multiple.

(3) Multiple lesions following sunburn (Poth, 1939), or gasoline burns (Ereaux, *et al.*, 1955). At the donor site of a skin graft (Brown and Fryer, 1955). Unknown cause (Grzybowski, 1950; present case).

(4) Multiple, but fewer and larger, with a familial incidence (Ferguson Smith, 1934).

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POTH, D. O. (1939) *Arch. Derm. Syph., Chicago*, **39**, 228.
SMITH, J. F. (1934) *Brit. J. Derm.*, **46**, 267.

Dr. J. Ferguson Smith: I agree largely with what Dr. Smith has said. We have not yet enough material to establish definitely the identity or otherwise of kerato-acanthoma and the multiple type which I described first in 1934. The present case seems to be one of the latter type. As has been explained, the lesions have not been left to develop and there is extraordinarily little now on which to base a diagnosis but in one or two of the biopsy sections there are those very isolated cell-nests, which are not quite like the growing edges of a kerato-acanthoma. If a large number of sections from both types are examined you will find many appear to be essentially identical but some of those from the multiple type will show changes which are not found in kerato-acanthoma.

The pitting in this man's face is characteristic of the multiple type, and, if not absolutely diagnostic, is at any rate highly suggestive. These cases do not usually behave malignantly, but they may do. One or two authors who have published series of kerato-acanthoma have included cases which turned out to be truly malignant and the father of one of our multiple cases, who himself was said to have had numerous lesions which healed spontaneously, died of what was at the time called simply "epithelioma of the face". It may have been coincidental; it may have arisen on one of the self-healing lesions, as epithelioma may on any scar or site of chronic irritation; or it may have been a "self-healing" lesion which just forgot to heal. In my original patient, who died ultimately of meningitis, secondary to old otitis media, we found, *post mortem*, adeno-acanthoma-like metaplasia of a tumour at the anus, with deep infiltration of the sphincter by epithelial strands and cell-nests. In the amputated ear of another patient we found a perineural lymphatic space packed with epithelial cells, quite deep (Currie and Smith, 1952). Add to these findings occasional abnormal mitotic figures, and I suggest that if all this is not accepted as genuine carcinoma, we are left without any firm histological criterion of malignancy.

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Dr. Brian Russell: Most of the carcinomata have appeared on areas exposed to the sun in Egypt—his face, forearms, thighs and knees. In my opinion, squamous carcinomata of the skin are much more serious in some sites than in others, even though the histological appearances may be identical. Carcinomata arising in thin skin areas such as the vermilion borders of the lips, the helices of the ears, the external genitalia, and the backs of the hands are those which tend to ulcerate and metastasize early, perhaps because of the more rapid involvement of lymphatics in subcutaneous structures. Similarly, carcinoma arising from atrophic scarred areas of lupus vulgaris or lupus erythematosus is also, in my experience, likely to metastasize early. While some examples of that type of carcinoma known as kerato-acanthoma regress spontaneously, others may reach a stage at which, for some obscure reason, the process becomes irreversible and frank invasive carcinoma develops.

Dr. C. D. Calnan: We have recently seen at St. John's Hospital a brother and sister with lesions identical to these, especially the very small nodular ones. There was no other family history or Scottish ancestry, but the histology confirmed the diagnosis of Dr. Ferguson Smith's self-healing epithelioma.

The following cases were also shown:

Pityriasis Lichenoides Chronica et Varioliformis Acuta.—Dr. E. PRETTEJOHN (introduced by Dr. J. R. SIMPSON).

Hansen's Disease (slides and photographs only).—Dr. P. J. FEENY.

Pemphigus Foliaceus.—Dr. LOUIS FORMAN.

Lichen Pilaris seu Spinulosus.—Dr. N. A. THORNE.

Lichenoid Eruption with Nail Changes, Possibly Due to Mercury.—Dr. R. G. HOWELL (for Dr. G. B. MITCHELL-HEGGS).

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Section of Ophthalmology

President—R. C. DAVENPORT, F.R.C.S.

[*November* 10, 1955]

DISCUSSION ON THE TREATMENT OF KERATOCONUS [*Abstract*]

Mr. Frederick Ridley reviewed the records of 92 cases of keratoconus sent to the Contact Lens Department at the Westminster Branch of Moorfields, Westminster and Central Eye Hospital between 1951 and 1955.

The general picture of the disease as seen in this series is set out and the results obtained by the fitting of contact lenses are examined, both as to visual improvement obtained and wearing time. Allergy—hay fever, asthma, allergic dermatitis—is seen in 15% of cases, or 3·65 times the average for all other groups (2,000 cases in all). Lenticonus is present in 10% of cases. The 92 cases included 42 males and 50 females and they were similarly affected in all age groups. Right and left eyes are similarly affected as between males and females at all ages and the evidence does not suggest that the right or left eye is more frequently the first eye to be affected in either sex. The analysis suggests that the disease rarely commences before 12 or after 25 years of age, and that the peak incidence is at 16·1 years.

Keratoconus as a rule runs an acute course, vision deteriorating on the average from normal to 6/36 in the first eye to be affected, and to 6/18 in the second eye to be affected within three years of the case coming under observation. The improvement in vision obtained with contact lenses as compared with spectacles is often dramatic, many individual eyes seeing normally with contact lenses as opposed to less than 6/60 with spectacles. Not only do keratoconus patients fitted with contact lenses enjoy this great improvement in vision, but they are at the same time rendered iseikonic, fully binocular with a full visual field, and in the more severe cases are made much more comfortable physically.

Particular attention is drawn to the importance of keeping a close watch on the fellow eye from the first moment a case of keratoconus is diagnosed. Any shift of the total refractive error in the second eye, usually towards myopia, is presumptive evidence that this eye is already affected by the disease. Since the majority of these cases become bilateral and since the second eye seems to be involved within two years of the first in most cases this is a feasible clinical procedure which may assist in the discovery of the ætiology of the condition and may enable treatment to be instituted much earlier than is possible if evidence of visual deterioration with spectacles in the second eye is waited for. In only one case was evidence obtained for an eye deteriorating after a contact lens had been worn regularly, and it is very possible that the wearing of contact lenses arrests the progress of the disease.

Only two cases in this series have voluntarily given up wearing their contact lenses. 23 cases who have worn their lenses more than one year and up to four years are known to average thirteen hours of wear daily. It is concluded that contact lenses enable a high proportion of affected eyes to see normally, that the lenses are worn well and that the great improvement in vision as compared with spectacles, and the successful wearing times achieved—thirteen hours daily on the average—are likely to be permanent.

Mr. A. G. Leigh: The indications for keratoplasty in keratoconus are: (1) Thinning of the cornea and the development of ectasia which shows signs of bursting. (2) Failure of a contact lens to improve vision on account of the development of opacities at the apex of the cone.

The special difficulties encountered in grafting in cases of keratoconus are due to the softness and thinness of the recipient cornea. These difficulties can be overcome by controlling the pressure of the lower lid on the cornea in the post-operative period and lamellar grafting as a preliminary procedure to perforating grafting in cases of very thin corneæ.

The histological features of tissue removed from recipient corneæ were demonstrated.

Mr. Harold Ridley: Keratoconus is uncommon in this country where travel is easy and intermarriage of blood relations rare so that few British surgeons have operated on many cases.

In keratoconus corneal grafting is indicated on optical as opposed to therapeutic grounds and only good prospects of a clear graft can justify surgery.

Eyes with keratoconus would seem promising for grafting because the cornea is unvascularized and therefore provides a good bed for the graft and there are no intra-ocular complications. On the other hand difficulties are presented by gross and extensive thinning of the cornea and the abnormal shape of the anterior segment. The shape of the cornea may be improved by Castroviejo's method of applying a cautery to the cone to cause shrinkage before trephining. Though 7 or 8 mm. full thickness discs are preferable, severe cases, which alone justify surgery, may demand a 9 mm. graft to rectify the contour and avoid thin areas. Large grafts, however, risk peripheral anterior synechiæ and vascularization from limbal vessels. Numerous direct edge-to-edge sutures are necessary to regularize the contour of the anterior segment and these entail some damage to the periphery of the graft.

The results of my grafts which have been performed on severe cases of keratoconus only have been much less satisfactory than expected. It would seem that mild and moderate cases are safely and satisfactorily treated with contact lenses and that the risks of surgery should be accepted only in severe keratoconus where the cornea is breaking down.

Mr. B. W. Rycroft: It is of considerable interest to examine the efforts of our predecessors in their treatment of keratoconus. About 1747 the disease was recognized as an entity by Demours and the term "conical cornea" was applied to it by Wardrup in 1808. The glistening appearance of the apex of the cone had been noticed and likened by Pickford to a "dew drop" or "a piece of solid crystal" embedded in the corneal centre.

The cause of the defect was, and still is, obscure though many theories have been advanced. To me it has always seemed convenient to regard it as a form of "anterior myopia" due to structural weakness in the sclerotic coat of the eyeball. There is also no doubt of the familial tendency, being more common in women than in men, although in the Afrikaans group of the inhabitants of South Africa where the disease is very common the familial tendency seems to have an equal sex distribution.

The diagnosis is generally easy but it must be carefully differentiated from early keratoglobus and early progressive myopia: a distinguishing feature is the even thickness of the cornea in the two latter conditions.

Previous treatment, in the absence of the exquisite optical aids which we possess to-day, was necessarily surgical and this is not surprising when we remember that towards the latter part of the Nineteenth Century the ophthalmic surgeon had recently acquired the advantages of general anæsthesia and conceptions of antisepsis. Moreover, his tools were beginning to take on the familiar appearance of those in use to-day.

Surgical manœuvres such as removal of the lens (1817), displacement of the pupil (1840), removal of the conical apex (1845) need only be mentioned. About 1869, however, there was considerable interest throughout Europe in the set of trephines which Bowman had constructed and which he used to excise the apex of the cornea down to Descemet's membrane: no doubt he was influenced by the ideas on lamellar keratoplasty which at that time were seeping through Europe. But in spite of all these surgical manœuvres no satisfactory results were obtained and the position was summarized by Demours when he wrote "je consais de n'y rien faire de particulier," and Lawrence emphasized this by saying "if we do not know the cause of the affection we cannot be prepared to propose any rational treatment: I cannot say that I have seen any plans productive of benefit".

The picture to-day is vastly different for conical cornea is a curable disease and is no longer a serious threat of blindness. The choice is between the use of contact lenses and keratoplasty.

My practice has been to use a corneal graft where there is extensive scarring of the corneal apex and where contact glasses do not improve vision to useful limits. With the improvement in the fitting of contact lenses the cases of keratoconus which come to surgery have sharply diminished and no case of conical cornea should have an operation until expert advice on contact glasses has been obtained. Surgery should be reserved for those cases where there are many splits in Descemet's membrane and recurrent attacks of keratopathy which in the old days would be treated by apical cautery and optical iridectomy.

At East Grinstead we have received valuable assistance from the Contact Lens Department at Moorfields under the direction of Mr. Frederick Ridley and in our last 17 cases 10 have good tolerance of contact glasses and lead normal lives. 4 are limited to about four hours'

tolerance with which they are well satisfied: 2 have been operated on by corneal graft surgery and a third has had a corneal graft operation by Professor M. Amsler.

Undoubtedly the results of surgery in keratoconus are brilliant as seen in the latest figures (1953) of Townley Paton who reports 84 cases or 89·3% with clear grafts and in the later series of Fine who reported 70% of clear grafts in 37 cases. The reason why corneal grafts are particularly successful in keratoconus is because the donor graft is entirely surrounded by normal corneal tissue.

My summary of these cases would be that contact glasses should be tried in every case but that a corneal graft should certainly be carried out if good toleration with contact glasses is not obtained and if visual improvement is not satisfactory.

[*March* 8, 1956]

Implications of the Vascular Anatomy of the Optic Nerve and Chiasma

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The vascular anatomy of the optic nerve has been much investigated and there has been conflict of opinion on several points which are of potential clinical significance. The results of an investigation into the blood supply of the optic nerve and chiasma have recently been reported (Steele and Blunt, 1956), and it is the purpose of the present paper to present a brief résumé of this work and then to review, in rather more detail, some of the issues which have been the subject of past controversy and to discuss some of the implications of the blood supply of the optic nerve and chiasma.

Blood supply of the optic chiasma (Fig. 1).—The optic chiasma is supplied, on its inferior

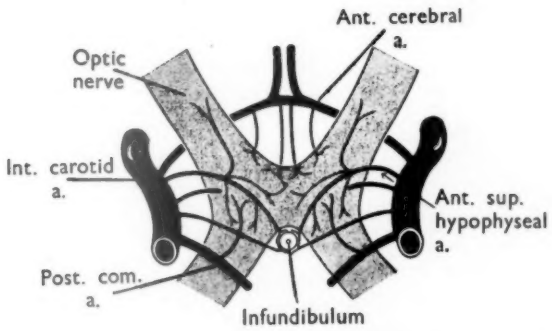

Fig. 1.—The blood supply of the optic chiasma. Diagrammatic.

surface, by branches of the posterior communicating artery, by a direct branch of the internal carotid artery, and by the anterior superior hypophyseal branch of the internal carotid artery which also sends a recurrent branch along the under-surface of the intracranial part of the optic nerve. On its superior surface, the chiasma is supplied by branches of the anterior cerebral and anterior communicating arteries. All the feeding vessels ramify in an overlying pial plexus.

The venous drainage of the inferior surface of the optic chiasma is provided by venous networks which are connected by transverse branches and which drain into the basal veins. The veins from the superior surface of the chiasma drain into the anterior cerebral veins.

The capillary plexus of the chiasma is of uniform density; it is fed by branches from the overlying pial plexus (Fig. 2). The most striking feature of the intrinsic vascular anatomy of the chiasma is the free intercommunication which exists between the capillary plexus of the chiasma and that of the hypothalamus.

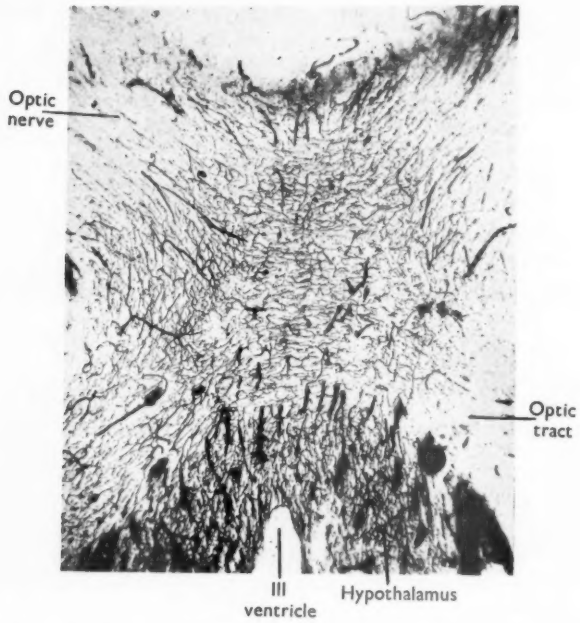

FIG. 2.—Oblique section of optic chiasma. Sodium nitroprusside-benzidine. 200$\mu \times 10$.

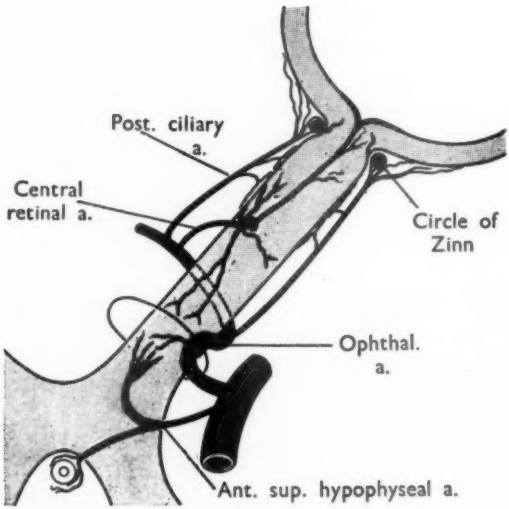

FIG. 3.—The blood supply of the optic nerve. Diagrammatic.

Blood supply of the optic nerve (Fig. 3).—The intracranial part of the optic nerve is almost entirely dependent for its arterial blood supply on the recurrent branch of the anterior superior hypophyseal artery: twigs from the ophthalmic artery also feed the pial plexus close to the optic foramen.

The pial plexus over the intra-orbital part of the nerve receives branches from the ophthalmic artery, from long and short posterior ciliary arteries and from the arterial circle of Zinn which links the short posterior ciliary arteries in the duro-scleral junction. The central artery of the retina also contributes branches to the pial plexus before entering the optic nerve: whilst in the nerve substance it gives off branches which feed the intraneural capillary plexus (Fig. 4).

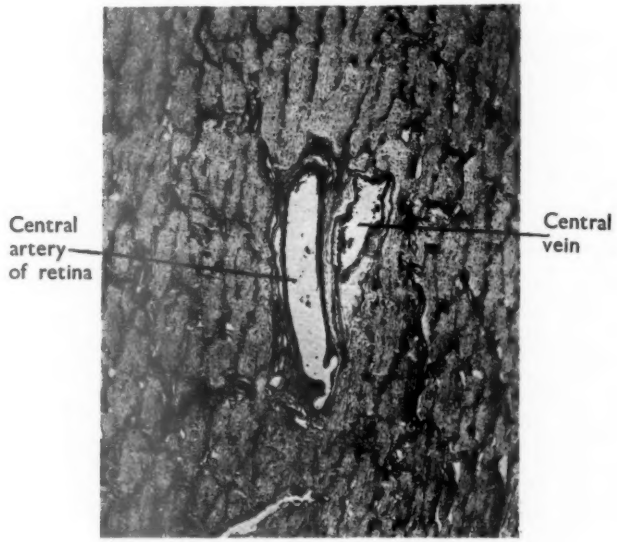

FIG. 4.—Horizontal L.S. of optic nerve. Hæmatoxylin and Biebrich scarlet. ×32.

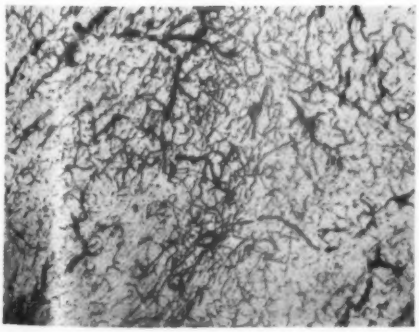

FIG. 5.—Horizontal L.S. of intracranial part of optic nerve. Sodium nitroprusside-benzidine. 200μ × 28.

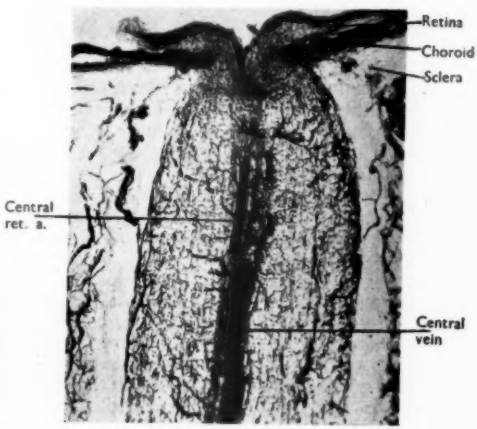

FIG. 6.—Horizontal L.S. of optic nerve. Sodium nitroprusside-benzidine. 200μ × 11.

The venous drainage of the part of the optic nerve behind the entry of the central retinal vein is mainly into neighbouring venous channels, though, in the intra-orbital part of the nerve, some vessels run distally to enter the central retinal vein. In the intraneural part of its course this latter vessel receives numerous tributaries which provide the main venous drainage for the anterior part of the optic nerve (Fig. 6).

The intracranial part of the optic nerve has a rich capillary plexus continuous, on the one hand, with that of the optic chiasma, and, on the other, with that of the intra-orbital part of the nerve (Fig. 5).

The capillary plexus of the intra-orbital part of the nerve is also extremely rich (Fig. 6). In the lamina cribrosa, where it becomes even more dense, it is in continuity with the retinal capillaries anteriorly, and, at the margins of the lamina, with the choroidal vessels.

Discussion

The optic chiasma and intracranial part of the optic nerve.—The main problems in regard to the blood supply of the optic chiasma and intracranial part of the optic nerve would seem to be whether the vascular arrangements are dominated by any particular vessel, and whether there is any possibility of the existence of autonomous zones of blood supply which might render the nerve liable to ischæmic damage from a focal arterial lesion.

The gross arterial blood supply.—Magitot (1947) claimed that the blood supply of the intracranial part of the optic nerve is totally independent of that of the intra-orbital part. Dawson (1948), however, was of the opposite opinion for he considered that the main blood supply of the intracranial part of the nerve was from a pre-chiasmal branch of the ophthalmic artery. In no specimen out of 34 nerves examined for this feature (Steele and Blunt, 1956) was such a vessel encountered: small recurrent branches of the ophthalmic artery did, however, supply the pial plexus and the blood supply of the intracranial part was not therefore independent of that of the intra-orbital part of the nerve. Direct branches from the anterior cerebral and anterior communicating arteries to the intracranial part of the nerve (Wolff, 1954) were not encountered; the anterior superior hypophyseal artery constantly provided its main arterial supply. In view of the fact that the same vessel provided branches to the optic chiasma which were more numerous and more far-reaching in their ramifications than the other arteries concerned, it seems possible, on purely anatomical grounds, that it may dominate the arterial blood supply to an extent which could be of practical significance.

The intrinsic vascular anatomy.—Although glial septa were found to be comparatively sparse in the intracranial part of the optic nerve, as was noted by Vail (1948), the paucity of septa was not mirrored in the density of the capillary bed (Steele and Blunt, 1956). No evidence was found to suggest that the capillary bed was denser on the nasal than on the temporal side of the nerve, as was suggested by François and Neetens (1956). This latter discrepancy in result may repay further consideration. Variations in apparent capillary density as shown by sodium nitroprusside-benzidine staining techniques are easily produced by the handling of tissues prior to fixation: blood may thus be squeezed from one part of the capillary bed into adjacent regions. Since François and Neetens do not state whether they found a less dense capillary bed on the temporal side of the nerve on more than one occasion, an artefact might well explain their findings.

It is suggested that there is no evidence from the capillary arrangements which would favour any suggestion that these regions might be more liable than others to the effects of ischæmia (Vail, 1948). There is, moreover, no evidence that the vascular anatomy of the intracranial part of the optic nerve is such that the papillomacular bundle might be more specifically exposed to the effects of ischæmia than the remaining fibres, as was suggested by Behr (1935).

The intra-orbital part of the optic nerve.—Most of the controversy centred on the blood supply of the intra-orbital part of the optic nerve has concerned, directly or indirectly, the central artery of the retina. All investigators except François and Neetens (1954) are agreed that the arteria centralis feeds the pial plexus immediately prior to its entry into the optic nerve (Fig. 7; 1). Anterior and posterior axial branches derived from the extraneural part of the central retinal artery were described by Wolff (1954) (Fig. 7; 3). A posterior axial artery was described by Beauvieux and Ristitch (1924) (Fig. 7; 2). The existence of any such axial branches has been denied by François and Neetens (1954). They did, however, describe an axial vessel which, they stated, arises more proximally from the ophthalmic artery, and which they called the central artery of the optic nerve. Branches of the central retinal artery with intraneural origins and distribution (Fig. 7; 4) were described by Leber (1903), Abbie (1938), Wolff (1940) and Bignell (1952), and their presence has recently been elegantly demonstrated by Wybar (1956). However, the existence of such intraneural branches has been denied by Beauvieux and Ristitch (1924), Magitot (1947)

and François and Neetens (1954 and 1956). Anastomoses between branches of the central retinal artery, and the circle of Zinn (Fig. 7; 5) have been described by Leber (1903), Wolff (1940) and Wybar (1956), but their presence has been denied by Beauvieux and Ristitch (1924), Behr (1935) and François and Neetens (1954). There has thus been conflict of opinion on every detail concerning the distribution of the central retinal artery in the optic nerve.

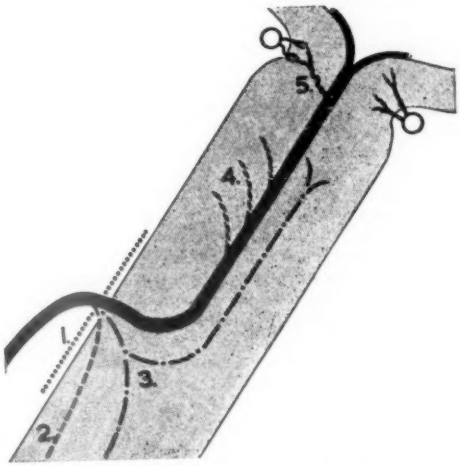

FIG. 7.—Previous observations on branching of central retinal artery.

Pial branches of the arteria centralis retinæ were constantly found (Steele and Blunt, 1956) and their terminal ramifications were noted to penetrate the nerve substance for a short distance only. In no specimen was there evidence of true anterior or posterior axial vessels or of a central artery of the optic nerve (François and Neetens, 1954), though injections were always made proximal to the described point of origin of the latter vessel from the ophthalmic artery. These findings confirm Wybar's observation that it is not possible to follow any axially directed vessel, other than the central retinal vessels, for more than a very short distance in the optic nerve. It is noteworthy, in this connexion, that François and Neetens (1954) do not record having injected both the central retinal artery and the central artery of the optic nerve in any individual specimen.

In all of 12 optic nerves examined by various techniques (Steele and Blunt) in which evidence of intraneural branching of the central retinal artery was specifically sought its presence was detected (Fig. 4). It is therefore suggested that the purely negative evidence on which such intraneural branches have been denied is based on failure to inject the central retinal artery adequately.

The presence or absence of anastomoses between retinal and ciliary systems of vessels is clearly a question of some practical importance. The extremely rich capillary bed of the optic nerve (Fig. 6) is fed, on the one hand, by vessels derived from the posterior ciliary arteries and the circle of Zinn and, on the other, by branches of the central artery of the retina. In the lamina cribrosa the capillary plexus of the optic nerve comes into continuity with that of the retina; at the margins of the lamina there is capillary continuity with the choroidal vessels, as was described by Leber. There is thus ample evidence of capillary anastomoses, but, in preparations stained by the sodium nitroprusside-benzidine technique (Steele and Blunt), no evidence of anastomotic vessels of more than precapillary calibre was found; in serial sections stained with hæmatoxylin and Biebrich scarlet, no anastomotic vessels with muscle tissue in their walls were encountered.

The dense capillary network which has been demonstrated at the lamina cribrosa (Fig. 6) contrasts with the observations of François, Neetens and Collette (1955) who injected the vessels with thorotrast and used a micro-arteriographic technique to examine the capillary bed. They reported that, as the papilla is approached, "the capillaries disappear". It is suggested that this finding was due to incomplete filling of the capillary bed since, in specimens stained by the sodium nitroprusside-benzidine technique (Steele and Blunt),

its great density was constantly apparent. The tissue of the optic nerve is relatively indistensible at the lamina cribrosa and this may explain the difficulty of examining its capillary bed by injection techniques; it may also explain why Leber's observation of a rich capillary anastomosis with the choroidal vessels has remained unconfirmed in later investigations in which injection methods only have been employed.

It may be noted (Fig. 6) that the retinal capillaries, as they run out from the lamina cribrosa, are heaped up in the papilla. It seems quite possible that a relatively low increase in intra-ocular pressure, occurring in cases of low-tension glaucoma, might compress the capillaries in the papilla and so impair the nutrition of the fibres at the nerve head. Moreover the blood supply of these fibres might well be further reduced by compression of the choroidal vessels against the scleral spur at the margins of the lamina cribrosa.

The rather rectangular pattern of capillaries in the intra-orbital part of the optic nerve behind the lamina is, in its main features, very constantly displayed by sodium nitroprusside-benzidine staining, but its appearance varies in details according to the amount of blood remaining in the vessels. Not infrequently the capillary pattern appears irregular and somewhat fragmented, giving an appearance which Cristini (1951) regarded as characteristic of glaucomatous atrophy of the nerve. The material on which the present observations are made was, however, from subjects in whom there was no known ocular disease.

From a general consideration of the vascular anatomy of the intra-orbital part of the optic nerve it seems possible to conclude that there is no evidence of any vascular autonomy. Axial and peripheral systems of vessels anastomose freely with one another, both in and behind the lamina cribrosa, by means of a rich capillary plexus. There is no evidence to suggest that the lamina cribrosa (Behr), or any other region, is dependent solely for its blood supply on a particular branch of the ophthalmic artery. It seems probable that the peripapillary part of the retina is supplied with blood, not only through the arteria centralis retinæ, but also through the retino-choroidal capillary anastomoses at the nerve head.

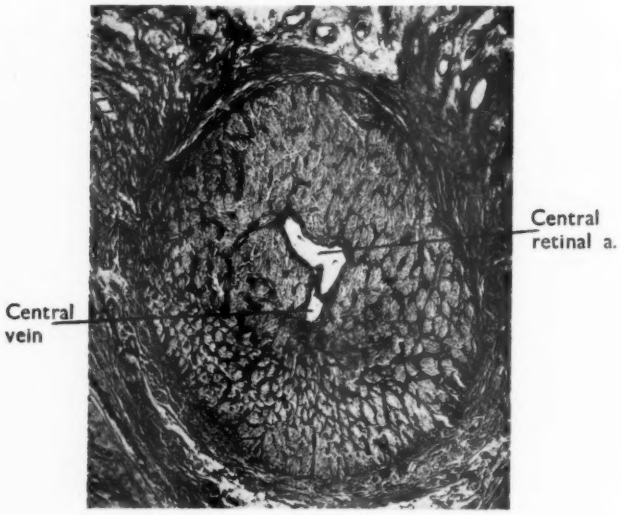

FIG. 8.—T.S. of optic nerve. Mallory. ×31.

Cilioretinal arteries.—Cilioretinal arteries are described as vessels which pass from the arterial circle of Zinn to supply localized areas of the retina: they curl around the margins of the optic disc and are noted most often on its temporal side. They are considered to be a common feature of ophthalmoscopic examination, from which their incidence has been given as 16·7% of 48 eyes by Lang and Barrett (1889), 14·2% of 120 eyes by Veasey and 8·7% of 439 eyes by Yoshida (Adachi, 1928), and "at least 25%" by Mann (1937).

In contrast to the above findings, in none of 72 optic nerves examined by various methods (Steele and Blunt) was there any evidence of a true cilioretinal artery. Such a negative observation is certainly to be interpreted with caution but it is, none the less, unexpected. Even more surprising is the fact that a perusal of the very extensive literature on cilioretinal

arteries has yielded reports of only 11 cilioretinal arteries directly observed. In 2 of these, noted respectively by Nettleship (1877) and Randall (1887), the arteries were found in nerves from patients with papilloedema and they therefore may have been compensatory collateral channels. In neither of the cases reported by Muller (1858) and Beauvieux and Ristitch were the origins of the apparently aberrant vessels observed. In Birnbacher's (1887) case the cilioretinal artery sprang from a vessel in the choroid. Another cilioretinal artery of this type was reported by Fuchs (1923), who found five cilioretinal arteries in all. However, the latter author himself states that "only exceptionally was it possible to follow the whole course" of the aberrant vessels, owing to the fact that complete serial sections were not available. A cilioretinal artery has recently been demonstrated by Wybar (1956), to whom I am grateful for the opportunity to examine unpublished photographs of the vessel. Its origin from a posterior ciliary artery was clearly defined and it might be said that this is the only firm evidence of a true cilioretinal artery, directly observed, which has been reported in the literature.

Although no cilioretinal artery was encountered during a study of 72 optic nerves (Steele and Blunt), bifurcation of the central retinal artery proximal to the papilla has been seen. In this instance (Fig. 8) the central retinal artery divided 0·4 mm. behind the papilla: the branch which left the parent vessel passed to the margin of the lamina cribrosa and then into the retina. Dissections of the arteria centralis and longitudinal frozen sections stained by the sodium nitroprusside-benzidine technique are not suitable material from which to determine the precise point of division of the central retinal artery, for the vessel is frequently displaced during dissection or sectioning. Premature division of the central retinal artery into its retinal branches was found in one out of five optic nerves which were available for critical study in this respect, a finding which is of interest in view of the apparent rarity of true cilioretinal arteries. On ophthalmoscopic examination it is not possible to see the origin of arteries considered to be cilioretinal and, even though such vessels may appear to curve around the margins of the optic disc, it is possible that they are usually examples of pre-papillary branching of the arteria centralis retinæ.

From another point of view, it has sometimes been assumed that the sparing of vision in part of the retina which may occur after embolism of the central retinal artery is due to the presence of cilioretinal vessels. Coats (1914), however, was of the opinion that this is not necessarily the case, and that the partial sparing of vision may be explained on the basis of the anastomosis between retinal and choroidal capillaries at the nerve head which was reported by Leber and of which confirmatory evidence has now been obtained (Steele and Blunt).

It is by no means claimed, on the basis of the available evidence, that cilioretinal arteries do not occur as normal variants in man, but it is suggested that they are not so common as appears to be the case from the results of ophthalmological examination.

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Some Clinical Problems of the Blood Supply of the Optic Nerve

By H. E. Hobbs, F.R.C.S.

The attention which has been given in recent years to the gaps in our knowledge of the blood supply of the chiasma and optic nerve and the communications which exist between this and adjacent areas of supply is of itself sufficient testimony to the need for more accurate anatomical information in the solution of the many unsolved problems which confront us here and Dr. Blunt's account of the work which he and Dr. Steele have been doing is both timely and welcome.

Detailed examination of the clinical problems in which the new knowledge of these vascular channels may be of importance is outside the scope of the present discussion, but there are one or two points of especial interest on which I should like to comment.

The first concerns the simple appearance of the optic nerve head as seen with the ophthalmoscope and the capillary network which Dr. Blunt's preparations have demonstrated so clearly and which must, in fact, be what gives the disc the appearance which we see when we examine the fundus. One's opinion of whether an optic disc is normal or abnormal is, of course, based upon a visual impression and it is common knowledge that as one's experience broadens one is less and less inclined to attribute abnormality to discs which show only small variations from the hypothetical normal, more especially when the evidence of accurate field studies is lacking. This impression is chiefly one of colour, a colour which has a definite character: one may be able to distinguish individual small vessels upon the disc, but the general appearance is that of a "blush"—I refer to it myself as the vascular filigree—which may be uniform over the surface of the disc or, more commonly, deeper on the nasal side than the temporal. I have found difficulty in reconciling this appearance with the accounts and illustrations of the blood vessels of the papilla in standard textbooks, although Wolff (1954) does refer, it is true, to the greater density of the capillary network in this area. Dr. Blunt's preparations of stained sections of the plexus here appear to me to demonstrate the luxuriance of this network in a way which agrees more nearly with the clinical appearance of the disc than any which I have previously seen and the density of the vessels here, intermingled as they must be with the closely packed non-medullated nerve fibres, must have particular significance.

The variations of the normal appearance of the disc are, as I have already mentioned, great; but most of us who use an ophthalmoscope at all frequently believe that we can detect here minor degrees of pathological pallor when they arise. I have myself been convinced of this only since, in 1946, it was my lot to examine the eyes of many hundreds of Air Force ex-prisoners returned from Japanese camps among whom a large group had the defective vision and central field defects characteristic of this particular type of nutritional neuropathy, and who showed on the temporal side of the disc a sector of pallor corresponding to the affected papillomacular bundle. However, the clinical problem of a doubtful optic nerve lesion in a patient whose responses are too uncertain for accurate field studies, either through coincident cerebral disease or congenital defect, is familiar to all of us; it is, unfortunately, often in just such cases that the detection of optic nerve damage may be of vital importance in providing localizing evidence of an intracerebral lesion to the neurologist or neurosurgeon. Closer attention to the capillary plexus on the disc by such means as the slit-lamp microscope and Hruby lens and by fundus photography may well provide us with more reliable methods of detecting optic atrophy in these cases: as far as the slit-lamp is concerned the examination is a direct and simple one; but photography of the disc at magnification great enough to be of value in this way presents technical problems which have yet to be solved. They are, I understand, already under consideration in the Department of Medical Photography at the Institute of Ophthalmology.

The distinction between such knowledge of a fundus picture and understanding of the essential disease process is one which has constantly to be borne in mind. This point was emphasized by Friedenwald (1947) in the 1946 Sanford Gifford Lecture when, dealing with the vascular changes seen in the retina, he remarked that the clarity of the clinical and pathological conception of this or that type of vascular disease is no longer paralleled in the idea of a characteristic fundus picture. He pointed out that the correlations which have been believed to exist between them tend to become merely statistical as, with the sharpening of classification, the number of atypical cases increases and he referred to the advantages of more exact methods of fundus examination in detecting the pathological changes present. He had in mind such methods as red-free light ophthalmoscopy and the use of the slit-ophthalmoscope.

Such considerations apply also to the changes seen at the optic disc and I have indicated a possible approach to simple optic atrophy in this way. The problem of correlating the changes seen at the nerve head in early simple glaucoma with the field defects and alterations

in intra-ocular tension suggests that more may be learned here, too, from a closer study of the disc. The diagnostic importance of pallor of the disc when cupping is doubtful is generally appreciated and has, of course, been emphasized by Duke-Elder (1940) and by Traquair (1942). It may be, as I myself believe, that the most satisfactory early evidence of the early changes of glaucoma is to be found as defects of the central field; but Traquair himself, stated (p. 131) that "the evidence of perimetry strongly indicates that it is the anatomical conditions of the disc . . . which determine the order in which the fibre groups suffer" and suggested that the explanation is probably anatomical and concerned with the vascular supply of the nerve fibres at the optic disc.

The manner in which optic atrophy, and subsequently cupping of the disc, arises in simple glaucoma is uncertain. According to the early classical views both result directly from the raised intra-ocular pressure commonly detectable clinically which, when produced *post mortem*, has been found to result in a herniation of the disc. The pressure was thus believed to act in a direct mechanical fashion and the theory took little or no account of its effect upon the vascular supply of the structures involved. Indeed, detailed knowledge of the blood supply of the nerve and papilla at this time would be considered incomplete by present-day standards. The contrary view, that both atrophy and cupping are attributable to degenerative processes in the nerve itself and that these are primary and independent of the intra-ocular tension, arose from the demonstration by Schnabel (1892) that in certain cases of glaucoma without raised tension the nerve behind the lamina cribrosa exhibited areas of cavernous degeneration comparable with those seen in other conditions of optic atrophy, and especially in that associated with gross arteriosclerosis of the intracranial vessels. According to this theory cupping is produced by the traction upon the lamina of the contracting degenerate areas of nerve tissue. Originally considered to relate chiefly to the group of cases referred to as "soft glaucoma" this hypothesis has attracted more attention of recent years: "spongy degeneration" in the nerve has been described by Loewenstein and Garrow (1945) as preceding the process of cavernous degeneration; Wolff (1947) has reconsidered the importance of the process in the genesis of glaucoma generally and attributed its effects to ischæmia of the nerve elements, and Cristini (1951), employing Pickworth's staining method in the examination of eyes enucleated in an advanced state of glaucoma, has detected a diminution in the number of vessels in the capillary network of the papilla and, to a less extent, of the retrolaminar part of the nerve, which he regards as the essential lesion of cavernous atrophy and as responsible generally for the manifestations of the glaucomatous process throughout the eye.

The capillary arrangements for the nutrition of the optic nerve fibres as they converge from the retina into the optic nerve are evidently of special importance in this matter: the nerve fibres themselves are here crowded together, especially on the temporal side where those of the papillomacular bundle occupy a large arc of the circumference of the disc and the superior and inferior paramacular fibres are further condensed into two massive bundles near the superior and inferior blood vessels at the upper and lower poles of the disc. Along this margin of the disc, therefore, there is a dense collection of nerve fibres, devoid of their myelin sheaths and totally dependent for their nutrition upon the delicate capillary plexus which surrounds them and which from its nature must be capable of very little resistance to increased tension within the eye. The present demonstration of this plexus, therefore, suggests to me a means by which quite small variations in the intra-ocular pressure may affect the nutrition of the fibres at the disc. This would be most likely to take effect first where the nerve fibres are aggregated close to the relatively rigid vessels at the upper and lower poles of the disc and might therefore account for the frequency of scotomata in the field-areas supplied by them. Sclerosis of the arterioles contributing to the capillary network would, naturally, contribute further to the ischæmia; but it seems likely to me that pressure alone, in these anatomical conditions, could be responsible. This view, of course, is essentially that put forward by Lagrange and Beauvieux in 1925. The intra-ocular pressure was regarded by them as initiating cupping in this way which of itself produced further compression of the small vessels in the anterior part of the nerve and led to further atrophy. The theory at that time, it will be remembered, was criticized on this ground because it was then thought that no nutritive branches were contributed to the nerve from the central artery. However, it appears from recent work, and Dr. Blunt's studies provide confirmation on this point, that such branches do exist and such a criticism need no longer, therefore, be considered valid.

Such a mechanism would explain the transience of the arcuate scotomata in many cases, their disappearance under miotics and their halting or even regression after decompression operations. Vascular sclerosis, as I have suggested, could further contribute to the damage and may well be the decisive factor in the elderly patients whose field loss continues when the intra-ocular pressure has been reduced.

Clinical evidence of the ways in which the fibres *within* the optic nerve itself are involved in toxic processes and by demyelinating disease presents difficult problems of interpretation in terms of the anatomy of the nerve. Certain toxins appear to exert a selective action upon the nerve fibres: the tobacco—methyl alcohol—lead and carbon disulphide group affect principally those of the papillomacular bundle with the production of centro-cæcal scotomata; whilst with quinine, salicylic acid, filix mas and certain organic compounds of arsenic a general depression of the field with peripheral contraction is usually seen. How these substances act is not clear: whether primarily upon the nerve fibres or by constriction of their blood supply; and why compounds such as optochin and quinine, which are apparently close chemical relations, should produce opposite effects in this sense remains a problem for biochemical research. The differing forms of nerve involvement in demyelinating disease, with scotomata which are sometimes quite disproportionate to the visual loss and which vary widely in their position and clinical course, are not all easily explained on the simple assumption that they result from plaques of differing size in this or that part of the nerve.

Accurate knowledge of the intimate details of the blood supply of the nerve is an obvious prerequisite of further research and the picture with which recent work now presents us, of a regular box-like capillary network throughout the nerve, rather more regular in its orbital and intracanalicular parts where it receives contributions from the central retinal artery than in its intracranial part, does a good deal to clear the anatomical air in this respect. Through this network the vulnerable papillomacular bundle, as it passes from the temporal side of the nerve at the disc to reach the centre in its posterior part, would appear to be supplied with blood in a uniform fashion with the rest of the nerve and not to receive a preferential supply which the believed existence of a central artery of the optic nerve might suggest. François, Neetens and Collette (1955) although they describe such a vessel would, I believe, be in agreement with this contention since they refer to the artery as forming a regular contribution to the pentagonal network of capillaries. This may well be relevant to the problem of toxic amblyopia since without such a special blood supply to the macular fibres in the posterior part of the nerve it is rather less easy to regard the amblyopias characterized by a central scotoma as due to ischæmia of these fibres and the conception of a toxin acting directly upon more highly specialized fibres is more readily acceptable. In the second group, where depression of vision is more general and where retinal vasoconstriction is known to be associated with the action of the toxin in several cases (quinine, filix mas, salicylic acid and ergot) a vascular mechanism seems more likely. Much of the effect of some of these substances is known to fall upon the ganglion cells of the retina; but the separate degenerative changes which have been described in the nerve indicate that this also suffers and it would seem more likely in this case that generalized constriction of the arteries of supply of the capillary network described would result in uniform ischæmia of the nerve with a general depression of the visual function such as is seen in these cases.

The manifestations of retrobulbar neuritis are so varied in their onset, the depression of vision which they cause, their course and prognosis that it seems almost certain that factors in addition to the position of the lesion in relation to the optic disc and the macular fibres must be held responsible in many cases. However, in its classical form, with rapid loss of vision, a central scotoma, negative fundus findings and complete, or almost complete, recovery, it is evidently closely associated with multiple sclerosis and with a plaque involving the macular fibres. Whilst the origin of this plaque remains still one of the more obscure problems of neuropathology it is, perhaps, of interest to glance in passing at the theories holding thrombosis or vasospasm responsible which have been put forward by Putnam (1933) and by Franklyn and Brickner (1947) respectively. Whilst either of these might seem to be at least an anatomical possibility if a special artery of supply to the central fibres of the nerve were present neither would seem to be likely with the capillary system which has been demonstrated to be present. On the other hand such a system would appear to be particularly vulnerable to the pressure of generalized œdema in the allergic condition which has recently been suggested as responsible by McAlpine, Compston and Lumsden (1955).

It is perhaps surprising when one considers the unequivocal nature of the clinical evidence for the existence of cilioretinal arteries that so few should have been directly examined *post mortem;* but although the present investigation demonstrates the presence of only capillary anastomoses between the retinal and ciliary arteries (through the circle of Zinn), the fact that in two out of thirty specimens the central artery itself arose from a posterior ciliary artery, taken with Wybar's recent observation of a retinal artery arising in common with a long posterior ciliary artery, indicates that there exists a certain degree of variability in the channels through which blood reaches the retina and optic nerve. This is not surprising when one considers the mode of development of these vessels from the primitive blood islands and vascular plexuses of the orbit and, indeed, when one recalls the numerous small vessels which one sees in normal eyes running from the region of the disc toward the macula.

Some of these possibly represent true cilioretinal vessels; but others certainly originate from the central artery itself and represent a macular blood supply separate from the main branches of the retinal artery, as the following case illustrates.

A man aged 61, under the care of Dr. Una Ledingham for severe hypertension, on January 23, 1956, experienced a sudden loss of vision in the right eye. When he was examined in Dr. Ledingham's clinic shortly afterward his vision was reduced to light perception, the retinal arteries were contracted and there was œdema of the retina. Subsequently there was a gradual recovery of vision and when I first saw him on February 3, he had 6/60 vision, persisting œdema at the macula and a central scotoma. I concluded that he had had severe spasm of the retinal artery and advised him to carry amyl nitrite capsules for inhalation if it recurred. On February 24, vision had recovered to 6/9 and, apart from some narrowing of the retinal arteries the retina showed little abnormality. On February 29, on his way to Dr. Ledingham's clinic, vision again suddenly failed in the right eye and he inhaled amyl nitrite with some improvement of vision. He was admitted to hospital at once, given 50 mg. of Ronicol by mouth and treatment with heparin intravenously was begun. By March 5 there had been considerable further improvement of vision; he could count fingers, his peripheral field had recovered to some 30–50 degrees but the central field showed a large absolute centro-cæcal scotoma. This somewhat curious finding was explained on examination of the fundus, for although the retinal arteries were very narrow they appeared to contain blood and again, at the macula, there was an area of œdema. The optic disc was pale and arising from the main trunk of the retinal artery could be seen a small branch running toward the macula which, in its proximal part was filled, even distended with blood; but, as it approached the œdematous area, suddenly became threadlike and disappeared. Evidently this is a separate branch supplying the macula which, having been occluded by spasm, has failed to reopen in its distal part as the rest of the retinal arteries appear to have done, at least to some extent.

The case bears some obvious resemblance to the reported by Wybar (1956) in which rather more improvement was seen after immediate oxygen therapy under Ainslie's care and indicates, I believe, the value of a more hopeful attitude to these cases in which at first sight irreparable damage to the retina appears to have been done.

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The President said that he supposed every ophthalmologist would insist that a cilioretinal artery is not uncommonly seen ; at any rate a vessel arising from the circle of Zinn behind the point where the central retinal artery is commonly obstructed and keeping viable a small area of temporal retina usually including the macula. Dr. Blunt's paper seemed to destroy some of the explanations which a central artery in the nerve, as described by François, could have given for clinical problems such as toxic neuritis, even though one allowed for the idiosyncrasy factor in the effect of these toxins.

Mr. Kenneth Wybar agreed that the demonstration of branches from the central retinal artery within the optic nerve depended to a certain extent on the thoroughness of the injection technique. He was most interested in Dr. Blunt's views on the possible rarity of true cilioretinal arteries. It might be that some of the so-called cilioretinal arteries which were seen ophthalmoscopically at the margin of the disc were in fact derivatives of the central retinal artery in the deeper part of the optic nerve head. He had found, however, a true cilioretinal artery in two neoprene casts, although unfortunately in one cast the connexion of the artery with the circle of Zinn was lost during the final stages of the dissection. In any case from a clinical point of view it was convenient to accept the concept of a cilioretinal artery to explain the occasional retention of some macular vision despite occlusion of the main stem of the central retinal artery.

Mr. P. D. Trevor-Roper asked what Mr. Hobbs felt to be the actual cause of the ischæmia that showed itself as a pallor of the optic disc in such lesions of the optic nerve and chiasma.

Mr. J. A. E. Primrose said that he once saw a patient with a very white disc but there was no defect of vision. It was not excavated but looked a wet disc. He thought it was a little œdematous. In one patient (shown at the meeting), although the veins were engorged the discs were not pink. He thought that might have been from œdema so that the ordinary colour of the disc vascular processes was a little patchy and the absence of colour was not an indication of atrophy.

Mr. E. F. King asked if there was a satisfactory anatomical explanation for those curious cases of temporal arteritis in which profound loss of vision occurred, often in both eyes, with relatively slight pallor of the optic discs.

Dr. Blunt, in reply, said that he would like to emphasize that the evidence did not suggest that cilioretinal arteries do not occur in man. It did, however, appear highly probable that many vessels which were classified as cilioretinal were really examples of pre-papillary branching of the arteria centralis retinæ.

It would not be possible to offer any anatomical explanation for a purely vascular lesion resulting in pallor of the optic disc in cases of aneurysm of the internal carotid artery unless, perhaps, the pallor was due to a partial occlusion of the ophthalmic artery. So many branches of the ophthalmic artery supplied the optic nerve between the chiasma and the lamina cribrosa that it appeared most unlikely that the phenomenon could be explained by occlusion of any particular branch of supply to the optic nerve.

It was indeed difficult, on the basis of the peripapillary anastomoses at the nerve head, to explain sparing of the macula alone after embolism of the central retinal artery. It could, however, be a possible explanation in some of the cases where macular sparing occurred that the block in the central retinal artery was situated anterior to the origin of a pre-papillary branch of the arteria centralis. In other cases, small vessels appearing at the temporal side of the disc, in association with sparing of macular vision, might be compensatory collateral channels developed from a peripapillary network which had been sufficiently rich to spare the macular region in the initial stages after the embolism. It was not therefore considered that the presence of vessels at the temporal side of the disc, found after embolism of the central retinal artery in association with a sparing of macular vision, could be advanced as evidence for the existence of true cilioretinal arteries, although, on rare occasions, such vessels might well be cilioretinal arteries.

Mr. Hobbs, also in reply, wished to make it clear that he did not think that the appearance of the disc alone without any other examination was an accurate indication of what was going on in the optic nerve. He was no less puzzled than others by the variations in its colour with optic atrophy and the case which Mr. King had mentioned was one which he had found especially difficult in this respect. One saw these cases of temporal arteritis with severe loss of vision and little or no pallor for a long time afterwards; although he could not be certain from his own experience that it did not ultimately appear. He wondered whether some explanation was not to be found in the fact that the intracranial part of the nerve was almost completely dependent upon the recurrent branch of the superior hypophyseal artery, so that occlusion of this vessel might abolish the function of the nerve whilst leaving the appearance of the disc more or less normal through the persistence of the intact capillary plexus at the optic disc.

With regard to Mr. Trevor-Roper's point concerning the relationship between pallor and nerve atrophy he felt that where lesions affected the blood supply at the disc this was probably fairly direct since here the closely mingled nerve fibres and capillary plexus were relatively unprotected and obliteration of vessels leading to nerve atrophy could be expected to result in corresponding pallor. The pallor which was seen after lesions farther back in the nerve was less easy to understand; but presumably the blood supply dwindled when its function became redundant through descending atrophy of the nerve fibres.

Section of Physical Medicine

President—J. SHULMAN, M.B., Ch.B.

[*January* 11, 1956]

DISCUSSION ON INJURIES IN SPORT

Dr. W. A. Fell: To start with, treatment for sport injury should be prompt, as hours saved at the beginning of disability may mean discharge days earlier. Hence the importance of having an "open department" for this type of case; though my own department is "closed" for all other cases, for athletic injuries the local practitioners know that it is wide open.

Cambridge possesses a University Health Centre for its undergraduates. Its Director, Sir Alan Rook, is very keen that all athletes should use its facilities and has provided space where we can treat students. One of the physiotherapists attends between 4.30 and 7.0 p.m.; the university provides the equipment. It is an admirable arrangement which I can strongly recommend to anyone else working in a University town. Firstly it does not interfere with undergraduate working times. Secondly, when the physiotherapist has finished with the patient and he needs hard exercise for his muscles before letting him back on the field, he can go upstairs to a fine gymnasium and into the hands of a co-operative and intelligent gymnastics instructor, who knows what "toughening up" means. Thirdly, it gets the undergraduates away from the hospital atmosphere to one where they can emulate each other in rapid recovery. The only disadvantage is that of difficulty in staffing at the beginning and end of term.

Any long lists showing the incidence of various types of injury would be fallacious in that they would not give an overall picture. If the injury necessitates bed rest, the patient is put into his College Sick Bay and will only attend at the Hospital when he is ambulant. Perhaps we ought to start in the Sick Bay but so far we have no means of doing so. Nor do we get all the injuries. Some are sent for private physiotherapy if they can afford it, and others are never likely to come near a Physical Medicine Department, e.g. most boxing injuries. We get almost exclusively injuries of the back and of the locomotor system.

Within this limited field I have come to the following conclusions. The most dangerous game appears to be Rugby football (this may be fallacious as more Rugger is played than Soccer). The injuries tend to be in the lower limbs—the knees, ankles, the muscles of the thigh, with the shoulders and the neck occasionally. Association football comes a close second with knee and ankle injuries predominating. Cricket is a long way behind with lesions of the shoulder (in bowlers), fingers (in batsmen and fielders) and an occasional low back strain or disc lesion. Athletics produces a surprising number of rather varied casualties, principally of the ankle and the tendo achillis. It always surprises me how many runners have a small tendo achillis, that is, small in cross section relative to the build of the rest of the body. Occasionally one gets sprains of the shoulder and elbow in discus and javelin throwers. Other sports give very little trouble; an occasional shoulder sprain in a tennis player, a teno-synovitis of the thumb tendons in a golfer, an osteo-arthritic flare up in an elderly elbow which takes too much hard ground with an iron club!

To revert to the football injuries. I have the impression also that sprains, whether of muscle or ligament, are more common in the first part of the first academic term, when after a summer's grazing these human horses are put to work again on the football field before getting absolutely fit. Later in the term when everyone is fitter the predominating injuries tend to be those of direct violence—the internal ligament of the knee, the shoulder dislocations and so on—rising to a peak when the intercollegiate competitions get under way in the middle of the second term. The moral would seem obvious, that it pays to be fit early on in the season.

I do not propose to deal in any detail with all methods of treatment. All sorts of procedures seem to have their advocates; a large number of undergraduates have had treatment elsewhere and are referred for continuation of previous treatment. Radiant heat, short-wave, massage, galvanism, ionization, procaine, Hyalase, hydrocortisone, all turn up, not infrequently in combinations which cancel each other out and which seem to be based on utter ignorance of the physiological effects of these physical procedures.

What are we trying to do? Surely when tissue is torn, wherever or whatever it may be, there is an extravasation of blood and excess tissue fluid in the region of the injury. This damps down movement and may in time organize into restricting adhesions. Therefore

it seems to me that any measure that will disperse this fluid and not allow it to reaccumulate is welcome, whether it be massage, exercises, Hyalase and so on. In these early stages the use of heat, should it go deep enough to reach the site of injury, will not do much to clear fluid away: it is more likely to increase the amount of swelling. I would plead for measures to try to prevent the swelling occurring, as by firm bandaging and the use of cold (again provided the lesion is near enough to the surface). We have the icebag and the ethyl chloride spray as used by the paratroopers but unfortunately we don't hold clinics at the playing fields! Both methods will have to be applied by the first-aid man whoever he is and both in my opinion are superior to any lamp. If I may be pardoned a wartime reminiscence, I was impressed by the routine treatment of patients with sprains and similar injuries in a hospital in India. They were brought straight from the game to the admission room, put to bed with an icebag, and started on non-weight-bearing exercises next morning, then discharged to light duty in two or three days, with a minimum of work for the joint affected. There, every ward had its big block of ice. Here ice is a rare mineral which has to be dug out of a fish shop.

What is the next step after the bleeding has been stopped and the swelling aborted? If there is torn tissue, surely time must be given for it to unite and heal. We cannot accelerate the rate of healing; we can only delay it by the wrong treatment. If we want union of torn tissue to take place the least we can do is to put the torn ends together and try to keep them there. Therefore I stress the importance of adequate immobilization applied in whatever manner is appropriate to the injury—splints, strapping, plaster and so on—as long as the immobilization will not allow a tension strain on the torn tissue. Just as stitches or clips hold a skin incision together so the technique used must hold the healing surfaces together. During the period of immobilization, relative or complete, massage and exercises, but no heat, will serve to disperse any swelling that may remain and when healing is well advanced harder exercises can be started so that wasted muscle can be built up again. Only in exceptional cases, when a muscle is inhibited by spasm or pain, should faradism be needed in an athlete.

This leads me to a very vexed question. An undergraduate is not very interested in the name of his injury. His first question is invariably "How soon can I play again?" I feel very strongly that we have to refuse to get him back on the field within two, three or four days or whatever period he wants and we have to tell him that he will go back when he is healed. We cannot accelerate that and before he subjects his once torn tissues to the severe strains of sport they must be soundly healed and his controlling muscles must be of good power. If not, then he stands the chance of repeating his injury and perhaps making it a great deal worse.

Yet, some people will say, there are methods such as the injection of local anæsthetic for relief of pain and restoration of seemingly normal function at once. Why not use them? I would say that they should be reserved for short duration emergencies only and never if undue strain is likely to be placed on the anæsthetized tissues. There are three reasons against their use. (1) Athletes are usually in their teens or early twenties. The muscles and joints that they own are the only ones they will ever get and they have to last another forty or fifty years. Do we want to accelerate the onset of degenerative joint disease? (2) In the more violent type of game there is a great chance of recurrence or exacerbation of the original injury. (3) A player's excellence or otherwise depends to a great extent on his co-ordination, and that in turn depends on his proprioceptive impulses. Is the co-ordination of an anæsthetized part going to be good enough to let him play well? Is an ankle, for example, which does not possess full painless movement going to be any use on a football field?

It is sometimes very difficult to make a decision. Too often a period off the game deprives the patient of a chance of a Blue, which means a lot—too much perhaps. I err on the side of caution. Sometimes I find my advice disregarded. If the outcome is disastrous, we must never say "I told you so!"

To sum up I would emphasize the following points in dealing with injuries: (1) The importance of accurate anatomical diagnosis of the injury, (2) the prevention of undue swelling by cold with or without firm pressure, (3) adequate immobilization and time for healing and (4) the maintenance of muscle power and its restoration speedily to its maximum.

Squad. Ldr. C. B. Wynn Parry: In the Royal Air Force an elaborate organization exists to provide all Servicemen with facilities for every type of sport. It is compulsory for everyone under a certain age to take organized exercise twice a week. The prime aim of physical training and sport in the Services is to keep everyone at maximum fitness so that their efficiency on the job is at its highest and also to help in the prevention of illness and injury.

It is axiomatic that fit people with good chest expansion are less likely to go down with respiratory disease—what is less readily accepted, indeed often overlooked, is that the

inculcation of strengthening exercises to specific muscles used in sport lessens the incidence of injury.

I wish to present some evidence that suggests that much more could be done to prevent sport injuries. I shall first show figures to give some idea of the magnitude of the problem. I shall then discuss some of the factors in treatment that we have found of paramount importance. Finally I shall try to show that Physical Medicine has an urgent contribution to make in the prevention of sport injuries.

In 1953 of all types of accident in the Royal Air Force, sport injuries accounted for 28%. Sport injuries were the fifth on the list of causes of time off duty throughout the Service. Many of these injuries become recurrent and may lead to permanent disability. For example 4% of all patients who have meniscectomies develop osteoarthritis of the knee-joint. Any patients requiring more than a few days' treatment in the Station Sick Quarters usually find their way to one of the Rehabilitation Units. An analysis of these figures will give an indication of the incidence of the more severe injuries.

Table I shows the percentage of all admissions that are due to sport injuries to the two Rehabilitation Units, Chessington and Collaton Cross.

TABLE I

6,118 Traumatic cases admitted to Medical Rehabilitation Units, Chessington and Collaton Cross. 1953–1955.
25·4% of all admissions are due to sport injuries.
27·4% are directly due to sport injuries.

Which sports are the most likely to lead to injury?

Table II shows the incidence in the four main sports played in the Royal Air Force,

TABLE II.—INCIDENCE OF INJURIES IN MAIN SPORTS IN ROYAL AIR FORCE

Soccer ..	84%	Gymnastics ..	4·7%
Rugger ..	9%	Athletics ..	2·3%

Amount of Soccer played is 2½ times amount of Rugger, but injury ratio is 9 : 1.

Soccer, Rugger, Gymnastics and Athletics. Although 2½ to 3 times as many people play Soccer as play Rugger, yet the ratio of injuries is 9 to 1. This is a significant difference and suggests that Soccer is more fraught with danger than Rugger.

Table III shows the incidence of the type of injuries in the four different sports and has been

TABLE III

Site of lesion	Soccer	Rugger	Gymnastics	Athletics
Knee	60% [1]	50% [1]	40% [1]	37% [2]
Lower limb (excluding knee) ..	27% [2]	17% [3]	33% [2]	47% [1]
Upper limb	10% [3]	25% [2]	12% [3]	4% [3]
Back	1·7% [4]	10% [4]	12% [3]	Nil

Small figures denote relative incidence within sport group.

compiled in order to find out if there are any particular types of injury more common to one sport than another.

Certain facts emerge from this table. First, the knee is obviously extremely vulnerable to injury in all sports. It is the first on the list in all except athletics, though even here knee injuries account for more than one-third of cases. Secondly back injuries are relatively uncommon. Thirdly, upper limb injuries are commoner in Rugger, particularly dislocation of the shoulder and fracture of the fingers.

When we come to consider the injuries more specifically we see from Table IV that tears

TABLE IV.—COMMON SPECIFIC INJURIES IN SOCCER AND RUGGER

Lesion	No. of cases Soccer		Rugger
Knee:			
Traumatic synovitis and cartilage tears ..	1,009		128
Torn ligaments	24		5
Lower limb:			
Fracture tibia and fibula	66		11
Fracture tibia	31	111	5
Fracture fibula	14		3
Pott's fracture	32		4

of the cartilages and traumatic synovitis are far and away the biggest cause of trouble. I have not attempted to separate these two as a patient with traumatic synovitis so often

develops the signs of a torn cartilage later that a distinction could not be in any way accurate.

However, it is seen that a fairly high proportion, 111 out of 1,176 (over 9%) sustain fractures of the bones of the leg.

It is interesting at this point to compare the types of injury caused by these sports with another type of sport, or so it is called by the young men who are its slaves—I refer to the motor cycle.

Table V shows the injuries that were treated in a three-year period that were directly due

TABLE V.—INJURIES SUSTAINED WHEN RIDING A MOTOR CYCLE
603 cases admitted 1953–1955 to both Units. Incidence 9·8% of all admission.

Fracture of bones of leg	147	Fracture forearm bones	42
Knee lesions	105	Fracture skull	7
Fracture femur	76	Complete right brachial plexus palsy	3
Fracture ankle	66		

to motor-cycle accidents. Again fracture of the bones of the leg and knee lesions were by far the most important injuries. Further evidence of the vulnerability of the knee is afforded by the fact that of some 1,100 cases of knee disability treated at Chessington in the last three years, 615 (56%) were caused by sport injuries and of 1,259 knee injuries at the two units due to sport, over 80% were caused at Soccer. At the Cadets Training College at Cranwell, sport naturally plays an important part in the curriculum and in a ten-week period September–November 1955 one-quarter of the Cadets received treatment for some form of injury sustained at sport, and 10% of these were knee injuries.

Finally in this broadside of statistics Table VI shows the injury admissions in 1953 through-

TABLE VI.—ROYAL AIR FORCE—1953
INJURY ADMISSIONS DUE TO ATHLETICS AND SPORTS
Fresh Cases of over 48 Hours

Nature of injury	Total No. of cases Due to "sports"	Total No. of cases All causes	% of cases due to "sports"	No. of cases per 1,000 strength due to "sports"
Dislocation without fracture ..	116	224	51·79	0·435
Sprains and strains of joints and adjacent muscles	1,611	3,145	51·22	6·043
Fracture of limbs	517	1,524	33·92	1·939
Superficial injury, contusion and crushing with intact skin surface	522	1,843	28·32	1·958
Fracture of skull (including bones and face)	132	484	27·27	0·495
Fracture of spine and trunk ..	32	153	20·92	0·120
Head injury (excluding fracture) ..	175	898	19·49	0·656
Internal injury of chest, abdomen and pelvis	22	187	11·76	0·083
Laceration and open wounds ..	124	1,104	11·23	0·465

out the Royal Air Force and the percentage due to sport in the various types of lesion. We are thus presented with the paradox that sport which is played to keep men fit is a potent cause of injury.

Treatment.—I will deal briefly with the treatment of two acute short-term injuries—sprained ankles and traumatic synovitis of the knee.

Sprained ankles are not as common as knee injuries in sport—but they occur often enough to require a concerted plan of attack if long periods of invalidism are to be avoided. Most of the sprained ankles in the R.A.F. occur during parachute jumping, which many of you perhaps would hardly regard as a sport, though the majority of those that have made jumps will tell you that they obtain a feeling of exhilaration which surpasses anything previously experienced, and it is quite easy to become an addict. It is vital for anyone injured on a parachute training course to get back on the course as soon as possible. Many are Territorials and cannot prolong their stay at the training unit. Hence we have a physiotherapy department at the school, as indeed we have at all the large training units in order to get the patient treated *at once*, intensively and on the spot.

Our regime is as follows: The limb is put in elevation, muscle spasm is relieved by massage to the whole limb but any tendency for the massage to be heavy leads invariably to perpetuation of the spasm. Active exercises are given the moment spasm is relieved and should progress to full painless range at the first treatment. A pad of 5/16 in. orthopædic felt is shaped to fit over the injured ligaments, windows being cut out to accommodate bony

prominences. A Scott Curwen bandage is now applied from the webs of the toes to just below the knee. Correct walking is then taught. Treatment is given twice a day.

When all symptoms have subsided and the ankle is objectively sound, the patient is put through parachute rolls from various heights until the physiotherapist is satisfied that he can progress to ground training. One of the most important features of this regime is to test the patient out thoroughly before return to jumping or sport so that therapist, patient and doctor are all confident of the outcome.

The average time from injury to return to jumping in a series of 48 cases has been six days.

Traumatic synovitis.—When a person twists his knee in a game of Soccer and an effusion develops, several things may have happened—a cartilage may have been torn, a ligament strained, or an acute traumatic synovitis sustained without other lesion. Whatever the lesion it is impossible to examine the knee properly and exclude any underlying damage until the effusion has subsided.

The quickest way of getting rid of an effusion is to put the knee in a back splint and restrict knee movements completely, until the effusion has disappeared. Throughout, the quadriceps must be religiously exercised by straight-leg-raising exercises, at least five minutes every hour.

Riseley-Pritchard (1955) working with Cranwell Cadets found that gross effusions disappeared in six to eight days and that return to full duty was quicker when the patient was at work in a back splint than if treated in bed. Millard and I found the same at Chessington in 1953.

The importance of vigorous, often repeated quadriceps drill cannot be over-emphasized. Table VII shows the difference in time under treatment at the Rehabilitation Centre following

TABLE VII.—EFFECT OF GRADE OF MUSCLE POWER ON TREATMENT TIME IN 1,723 CASES OF MENISCECTOMY

Grade of power on arrival	Average days at M.R.U.
29% poor	48·1
42% fair	36·7
29% good	29·1

meniscectomy between those whose muscle power was good, fair and poor on arrival. It takes three weeks longer to get a patient fit for duty after meniscectomy if his quadriceps are poor than if they are good on commencing intensive rehabilitation. For this reason the institution of exercises immediately is essential at the first treatment after a knee injury and as soon as the anæsthetic wears off after a meniscectomy.

Most professional footballers have given up the crepe bandage as a normal part of football gear—they have rightly seen that it weakens the knee, but far too often doctors and trainers put a crepe bandage on the knee and tell the patient to rest it till the swelling goes. Over and over again we have seen patients who have been treated in this manner and who have been left with permanent weakness of the quadriceps. They become much more liable to injury and the injury need only be mild to precipitate a serious disability.

Once a person has had traumatic synovitis, we believe that quadriceps exercises should be continued indefinitely if the patient is taking an active part regularly in sport. We advise all patients after meniscectomies to carry on ten minutes' straight-leg-raising exercises daily for at least a year—by which time it should have become a habit and they go on doing it for the rest of their lives. A photograph appeared recently in a newspaper with the caption "Bill Johnston is *getting himself fit again* after having a cartilage removed from his knee. Here Johnston . . . has his leg massaged during a game".

If only the newspapers could publish a photograph of a public sporting figure building up his quadriceps with *active* exercises, what a service they would do for Physical Medicine!

The treatment of severe long-term injuries is, of course, a vast subject. Here I should only like to plead for the establishment of more centres, residential or day centres, where intensive full-time treatment can be given. We have found that the full-time treatment at a rehabilitation centre not only reduces markedly the time off work but it can allow a much higher proportion of patients to obtain a better result. It is of supreme importance to start rehabilitation as early as possible. We encourage the start of rehabilitation treatment at the Centres within the first two to three weeks for a leg fracture. A patient with a fractured femur with a Küntscher nail can start non-weight-bearing exercises for hip and knee movements in the physiotherapy department and warm pool within ten days of nailing. The advantages of treatment of fractures in the early stages is the resulting prevention of limps, bad posture, wasted muscles and bizarre gaits.

The simple fractures of tibia and fibula return to full duty after an average period of four-and-a-half months after fracture on this regime.

It is very necessary that before returning to work and particularly to sport, the patient should test himself out thoroughly and ease himself gradually back to sport. One of the most potent causes of recurrence of disability and breakdown is the widespread tendency for patients to be frightened of using their limb after a fracture or operation and resting it, or not playing again that season. This is just as bad as charging into a Rugger scrummage a month after a bilateral meniscectomy, for all too often the patient rests the part only too literally and when he starts sport again it lets him down immediately. A great deal of propaganda needs to be done to ensure that vigorous active treatment is started as early as possible and intensively for all sport injuries and that patients should understand clearly how important their contribution is and that they must prepare themselves properly before starting sport again. It is just as important, though, not to prolong the period off sport a day more than necessary—for physical and psychological reasons.

After all the point of taking a cartilage out is so that the knee will be fit for duty in the office and on the sports field as soon as possible. We find so many patients who fear to use their knee for months after such an operation.

Finally and most important of all—prevention of injury:

I hope I have shown that the incidence of injuries in sport is high. Although the intensity of sport is perhaps greater in the Services than in civil life, yet the incidence of injury among professional footballers, Rugger players and in many other sports is high enough to cause concern. There are, of course, many factors to be considered—the state of the ground, the type of equipment used, the frequency of playing. Injuries are much more frequent when the ground is hard or frosty. Amateur teams and week-end players having a kick around suffer more from incorrect footwear and uneven grounds. But one thing that does seem to us to be quite avoidable is taking the field in a state of unfitness for the particular sport. It is obvious that general fitness such as is given by P.T. is essential—to develop stamina, vital capacity, alertness, speed and precision, but it does not seem to be widely accepted that the development of the specific muscles used in the particular sport will not only make for improved prowess but reduce the risk of injury. If a man wants to throw a discus farther than anyone else he does two things. He gets generally fit, by which I mean that he builds up his chest expansion, cuts down his smoking, goes for runs, and he also throws the discus thus developing the throwing muscles and he may well go through a comprehensive weight-lifting programme. It seems obvious that he is much less likely to damage his shoulder-joint or muscles when throwing the discus after training than when untrained. Transference of these principles to the realm of football does not seem, however, to be nearly as obvious to those responsible for the fitness of football teams. It is quite clear that the quadriceps is the footballer's muscle and if it is really strong the player can kick the ball hard, far and fast.

The knee-joint is also less liable to damage; that this is true is shown by the patient with gross laxity of cruciate or collateral ligaments. The only treatment for this condition is devoted quadriceps drill. But once the quadriceps is strong a patient with these conditions can often play football. When the quadriceps is weak, the mildest jarring causes the knee to give way. You will see now that I am advocating not only the regular daily continuation of specific remedial strengthening exercises after even the mildest injury but also the regular performance of those exercises as a prophylactic in all sportsmen. One criticism of this idea has been that this would tend to make the players conscious of injury, but players are conscious enough of the possibility and consequences of injury as it is. One or two professional teams include five minutes quadriceps exercises a day in their training programme. Most do no specific exercises at all.

We all know the dire consequences of enthusiasm untempered by preparatory training at the start of the Soccer and Rugger season and the line of plasters on the terraces of the Swiss ski resorts. It is only an extension of normal precaution to plead for specific exercises to prevent injury.

We hope to start an experiment in the R.A.F. next year in which specific strengthening exercises will be given to all fooballers in certain units and the incidence of injuries compared with those sustained in players who have not had the exercises. Thus statistical confirmation or otherwise in a large number of cases should be forthcoming.

It is interesting to note that in a newspaper last month appeared this paragraph:

"The Australian Board of Control may pay greater attention to the question of physical fitness of the players selected for the coming tour of England. This season Victoria instituted a course of physical training for its State squad. For a month before the first match a leading Melbourne P.T. instructor concentrated on the players and there was definite improvement in their physical condition. Board members argue that any expense involved in providing a physical course is infinitesimal compared with the long list of medical bills they have paid because of injuries caused by lack of sufficient preparation."

ACKNOWLEDGMENTS

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Dr. W. D. Fletcher: The injuries which primarily concern us in this Section are those of the soft tissues, so that our knowledge of the locomotor system should make us particularly adept in this field, especially if backed by a thorough knowledge of anatomy. Perhaps it is the absence of these two factors which causes such difficulty in diagnosis amongst Casualty Officers and House Surgeons. In fact, diagnosis is really an exercise in applied anatomy which I would like to illustrate by one example, the principle of which is already well known in Moseley's test for tears of the rotator cuff in the shoulder.

Whilst running a sudden pain is felt in the leg and there is pain on plantar flexion. Anatomical tests localize the trouble as being in the calf. Is it a rupture of a few muscular fibres, a ruptured plantaris or ruptured Achilles tendon? (They occur in this order of frequency.) The physical signs are almost the same since fairly powerful flexion is possible even in Achilles tendon rupture because of the peronei, long toe flexors and tibialis posterior. On the other hand flexion power is reduced in the other two conditions because of painful inhibition thus simulating a ruptured Achilles tendon. Pain is therefore abolished by procaine injection and the test shown in Fig. 1 applied.

FIG. 1.—Patient is asked to stand on tip-toe and the tension developed in the Achilles tendon is tested by direct pressure. After pain has been abolished by procaine injection tension is reduced only when tendon rupture has occurred as on the right.

FIG. 2.—Cyclist's palsy. Showing how pressure is exerted on the deep branch of the ulnar nerve as it passes between the pisiform bone and the hook of the hamate.

Dangers and their prophylaxis.—The most dangerous sports are the body contact variety, where some contact, often violent, or trial of strength with an opponent is almost inevitable even within the rules of the game, e.g. boxing, football, hockey, wrestling and lacrosse.

Boxing stands unique in these respects since the sole object is to injure one's opponent. Furthermore, one injury sustained in boxing is particularly dangerous since it is not often visible and often permanent. I am referring of course to the central nervous system. Groginess, traumatic asthenia, punch drunkenness and even hemiplegia occur all too

often, but what is more alarming is the interference with the higher intellectual function that can occur after only one bout.

Busse and Silverman (1952) examined 24 professional boxers in Colorado and found significant EEG changes in 9 which were more severe in those who had been knocked out. Larsson in 1954 reported changes in 13% of boxers following a bout and in 30% if they had been knocked down or out. Maitland (1928) estimates that nearly half of the professional boxers display a brain defect sooner or later.

Twenty years ago Carroll (1936) conducted a most thorough investigation into this condition and found that 5% of those who remain in boxing for five years become "punch drunk" and as many as 60% develop definite personality changes noticeable to their acquaintances. He stated "It is probable that no head blow is taken with impunity and that each knockout causes irreparable damage". The cause is probably repeated periods of cerebral anoxia but in those cases which have come to autopsy the changes resemble those seen in concussion. There are several clinical varieties.

Grogginess is a state of muscular flaccidity without loss of consciousness. He is said to be "out on his feet". It is in this state that severe damage can occur as the poor tone of the cervical muscles permits an abnormally wide range of movement of the head to occur at each blow.

Traumatic asthenia or asthénie chronique post-traumatique described by Boigey (1938) is characterized by a feeling of weakness, lack of initiative, fatigability and sometimes depression.

Punch-drunkenness is more widely known, being the subject of ill-placed humour in sketches, films and cartoons and in the profession is variously known as "punchy", "goofy", "slap-happy", "slug nutty" and "cutting paper dolls".

Concentration and memory is impaired and movements become slower and less co-ordinated. The knees give way more easily after a head blow and the subject remains dazed for a longer period. There is weakness of the legs, and dragging of the feet can be seen as he walks to his corner. The voice becomes thick although he is often voluble, sometimes with the euphoria of G.P.I. A form of automatism is very common where the sound of a bell will evoke pugilistic attitudes. This is often observed at the ringside of a fight where the old lag is seen mimicking the grimaces and attitudes of his colleague in the ring. It is less common in the more agile and intelligent, who receive fewer blows to the head and at the first sign of damage leave the ring.

Because of these effects, the Director of Physical Health Education of the U.S.A. recommended the abolition of boxing in 1938. In an editorial in the *Journal of the American Medical Association* in 1942, 87 known deaths were recorded, 47 in the U.S.A. It has been recommended that there should be (1) closer medical supervision, (2) research into providing a better glove.

Closer medical supervision is almost useless, as in order to be effective it would have to be of as high a standard as that described by Blonstein and Clarke (1954) in this country a year ago. But this is amateur boxing and such standards would not be practicable in the professional ring. Medical examination would prevent boxing by a man such as the Boston Tar Baby, who went blind in 1917 but was not stopped until 1924, when he was seen wandering round the ring trying to find his opponent, whom he had just knocked out. Or by Bill Softly who had diplopia and said, "Yes, I see two chaps in the ring and I hit the one that isn't there and the other one hits me".

As to research into a better glove, this in my opinion is valueless. Gloves protect the striker, and not the victim. In 1889 John L. Sullivan, the Strong Boy of Boston, boxed Jake Kilrain for 75 rounds and George Borrow and Conan Doyle described even longer bouts. The reason was that gloves were not used and damage to the hands limits the force of the blow. A special glove might completely eliminate cuts and metacarpal fractures but would still remain a human cosh and the kinetic energy transferred to the nervous system would remain the same. This can only be minimized by (1) abolishing the glove or using one only just thick enough to prevent cuts or abrasions, or (2) wearing a suitable head harness. Cornell University have devised a floor covering which cuts down injuries due to falling—this should certainly be used. There is now adequate medical evidence for the rest of society to weigh the moral value of boxing as against its dangers, to decide its future.

Football.—Injuries have been fully covered by the two previous speakers and whilst Association Football offers particular hazards to the knee, Rugby, being a body contact sport *par excellence*, produces the greatest number and diversity of injuries of any sport with the possible exception of ice hockey.

Prevention of accidents.—Minor muscle sprains and tears are most common at the beginning of the season, solely due to inadequate or unsuitable training: they practically

disappear if training can be started in August. Such injuries are more prevalent on cold days and on slippery or frosty ground. For those who are still doubtful about the significance of external temperature on muscle efficiency here is a field experiment all ready to hand. Thorough warming up by skipping or hopping in a track suit, even on a hot day, is extremely valuable in all athletics. For the same reason heated seats in the changing rooms of some clubs are of help in the period between changing and play. Consideration of these facts empirically discovered and applied in athletics should cause those who have despised the radiant heat lamp in recent years to have second thoughts.

As most injuries occur in the scrum or on contact with another player, these hazards can only be reduced significantly by changing the rules of the game. This has been brought about to some extent by Rugby League rules. Less weight in the scrum and fewer of them reduce these injuries and, furthermore, the ball does not remain "in play" after a tackle so that body kicks whilst the tackled player is on the ground do not occur.

Shoulder injuries can be minimized by O'Connell's (1954) suggestion of a ½ in. thick sorbo pad, 8 × 6 in., sewn to the inside of the jersey over the shoulder. Shin pads should be used more often. There is little doubt that a better boot would considerably reduce ankle strains and fractures, which are so common: the usual form is little more than a reinforced slipper which is rendered more unstable and a dangerous weapon by tri-nailed leather studs. The upper should be higher and of much more supple leather so that it can be fully laced at all times and give proper support to the ankle. Surely a more stable anti-skid device is possible than the present studs which are dangerous when on and equally so when detached as a cause of puncture wounds and lacerations. Tetanus is a definite hazard from such wounds and for this reason it is unwise to allow ground used for football to be accessible to horses at any time.

Ski-ing.—Here the most common type of fall is with the knee and ankle abducted and externally rotated.

In one series of 517 ski-ing injuries in the B.A.O.R. (Bruce *et al.*, 1953) a third were in the knee, a third in the ankle and a third wounds, blisters and due to miscellaneous causes. A hundred required admission to hospital and a third of all participants received some injury, mostly in beginners in their first three weeks of instruction. 70% attributed their injury to ski-ing off the hard prepared snow surface, or pista, on to soft snow, and only 10% to some special hazard such as a tree or rock. This information seems to indicate that beginners should (1) have instruction before commencing, (2) have training in ankle control with skis on rollers, and (3) avoid pista for the first three weeks.

There is one injury which I have seen most often after ski-ing, but also in other sports; it is particularly dangerous in that it appears trivial at first sight but can ultimately lead to severe and permanent disability due to knee stiffness. This is femoral detachment of the medial ligament causing para-articular ossification. The immediate treatment is firm compression over the medial femoral attachment to limit the hæmorrhage and thus prevent subsequent ossification from interfering with the function of the medial collateral ligament. Unfortunately, this treatment never seems to be given at winter sports, but on the other hand the cases are seen here three weeks later on their return home, which is the optimum time for recognizing this pernicious variety of tear. Tenderness, slight swelling and warmth, limitation of flexion (probably getting worse) at this time demands prompt immobilization until the activity has subsided.

Skill.—This is probably the most important single factor in reducing sports injuries and the lack of it was the reason for the greater proportion in the last series. Apart from the skill in any particular sport, there is also a special skill in avoiding injury in games, where even with the greatest mastery, a slight error of judgment can lead to a serious accident.

A good example is the high jumper—let us assume he weighs 10 st. and has jumped 4 ft. A mass of 140 lb. falling this distance and being brought to rest in ½ in. requires approximately 5½ tons upward pressure. This explains how serious fractures occur from an apparently innocent sport when the conditions are right for this force to be applied.

How is it avoided?

(1) The manner of the jump. If the legs are long and properly used the body need only move through a quarter of the distance. The potential energy is then reduced to 140 *g* foot poundals from 560 *g*.

(2) If he lands in soft sand and sinks 6 in. the force is now reduced to 280 lb. It is still twice his body-weight and sufficient to cause soft tissue injury.

(3) He does not keep the knee locked, however, and if he rolls sideways and applies the maximum body surface to the ground (say 2 sq. ft.) the pressure is reduced to 3 oz. per sq. in.

Having discussed how to stop, a word on how to start might be appropriate. Inefficient contraction of muscle leads to excessive force and tears. These are reduced by the use of a starting block.

Injuries peculiar to sport.—(1) Tibialis anticus syndrome (myosite du jambier antérieur) occurs in football and long distance runners (Oldfield, 1948; Hughes, 1948).

(2) Rupture of the oblique abdominal muscles and sometimes avulsion of the anterior superior iliac spines in javelin and discus throwers.

(3) Cyclist's palsy—this also occurs in parallel bar work and is due to pressure on the deep branch of the ulnar nerve, as shown in Fig. 2, as it passes between the pisiform and the hook of the hamate.

(4) Javelin thrower's elbow—fracture of coronoid and olecranon processes (Waris, 1953).

(5) Median and ulnar neuritis in javelin throwers. This injury is due to movements of wrist and elbow causing see-sawing of the nerves under the carpal ligament and with added bicipital aponeurosis when the median nerve is involved.

Treatment.—It is a pity that in the soft tissue injuries physical treatment often achieves little whereas it can achieve so much.

The correct methods of muscle development are too often not used and are of paramount importance, not only in prevention of injury, particularly of the knee, but in restoration afterwards.

Massage (I use the term in its widest sense to include any manual treatment) has for some time been neglected and consequently undeveloped or rationalized by us, who are after all the only people to do it properly.

No other medium can bring about the deep changes we require so effectively as mechanical means. For example, we are asked with monotonous regularity to give short wave to a hæmatoma. This is scratching the surface. If it is deep or near bone it is best left alone, if superficial or in a joint it should be aspirated, or evacuated through a small incision if necessary. If not accessible, it should be dispersed with massage—the area of absorption is then x times as great and will take $1/x$th as long to absorb and furthermore, no painful fibrotic mass or adhesion will remain to be a source of attraction for the procaine or hydrocortisone enthusiast. Procaine, whilst useful in diagnosis, has no place in the early treatment of soft tissue injuries, in fact, its use may convert a minor sprain to a major tear. Hyalase probably has a definite place, particularly in ankle sprains.

Splintage and pressure I regard as a mechanical means of minimizing reaction in the first few days which maintains the benefit achieved by cold in the first few hours. I never hesitate to use it on the score of joint stiffness.

Heat I have referred to and am quite happy with the superficial variety, i.e. wax and the radiant heat lamp.

Finally perhaps we should not dwell unduly on injuries lest the undoubted value of sport be lost. As Shakespeare has it in "The Tempest":

> "There be some sports are painful, and their labour
> Delight in them sets off: some kinds of baseness
> Are nobly undergone; and most poor matters
> Point to rich ends."

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Section of Otology

President—E. G. COLLINS, F.R.C.S.Ed.

[*December* 2, 1955]

DISCUSSION ON THE MANAGEMENT OF DEAFNESS IN THE YOUNG CHILD

Miss Edith Whetnall:

In spite of a popular misconception, otologists have been prominent during the last 150 years in showing that the hearing of a deaf child can, in fact, be trained (Ernauld, 1768; Pereire, 1768; Itard, 1802; Urbantschitsch, 1895). The method has never been generally accepted, I think, because of the lack of understanding of how the normal hearing child learns speech. The statement has just been made that it is not possible for the deaf child to acquire speech in a similar manner. What I would like to point out is that the child with normal hearing would not acquire speech under the conditions in which some deaf children are trained.

Figs. 1 and 2 show the hearing loss of two of the first children to be admitted to a London County Council Partially Deaf Unit. With this amount of hearing we would not now send

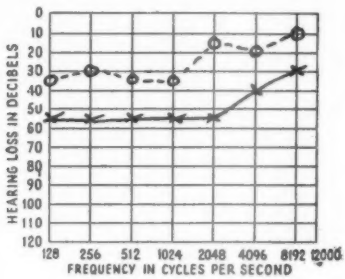


FIG. 1.—E. S.
Left x——x——x
Right o - - o - - o

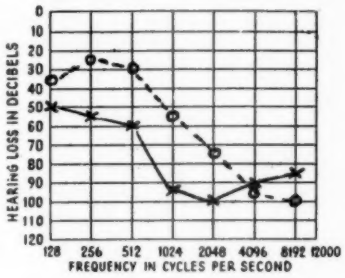
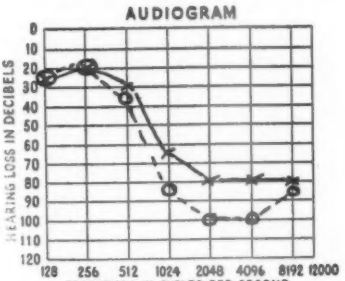


FIG. 2.—A. W.
Left x——x——x
Right o - - o - - o

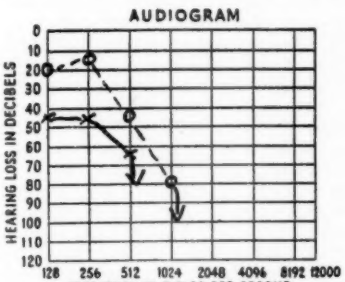
FIGS. 1 and 2.—Hearing loss of two of the first children to be admitted to a Partially Deaf Unit.

them even to a partially deaf unit, but at that time they were in schools for the deaf without hearing aids and being taught primarily by lip-reading. It is, in fact, possible for a child with a severe loss of hearing to acquire speech, given a little help at the right time. Sir Cyril Burt (Burt, 1952) long ago commented on the fact that now and again very severely deaf children were found in ordinary schools with good speech, in spite of having received no special training. (Fig. 3 shows the audiogram of a child belonging to this group.)



Left x——x——x
Right o - - o - - o

FIG. 3.—V. B. Child in ordinary school with no specialised training.



Left x——x——x
Right o - - o - - o

FIG. 4.—Child with severe high-tone loss, aged 8½ years. Holding her own in an ordinary hearing school. Her mother trained her hearing by talking into her ear.

A great deal is said about the so-called essential frequencies, i.e. those that must be present if speech is to be learnt through hearing. The audiogram shown in Fig. 4 is that of a child who lacks a wide band of those "essential" frequencies and yet has learnt to speak. I studied a group of children of this sort and found that in every case what had happened was that the mother had suspected deafness when the child was very young, often during the first year of life, and had talked into the child's ear. These cases showed that at any rate some severely deaf children could acquire speech through hearing. In fact the young child can learn to discriminate between any sounds, provided they are presented often enough and loud enough. He must receive a code of sound signals in association with their meanings at the right age in order for him to do this (Whetnall, 1955).

In contrast to this case is that of a child first seen at the age of 2 years. The parents admit suspecting deafness at 6 to 8 months because there was no response to their voices. Their first child had responded during the first month of life so that it is probable that they actually suspected deafness before 6 months. Even then they waited until the child was 1 year old before seeking advice. A severe degree of deafness was diagnosed and the child was referred to a clinic where unfortunately the value of auditory training was not understood. A Medresco hearing aid was issued. These aids are impossibly heavy for a child of this age but the parents were advised that the child was too young for a Monopack. Instruction was given almost solely in lip-reading, the hearing aid being used for a few odd half-hours each week. The parents were told that the hearing was of little value. As a result the child did not like the aid. The effect of concentration on lip-reading is to condition the child to ignore sound. Even so, at the first visit to me he wore a Monopack aid, with delight, for an hour. He responded to his name at 3 ft. and to a drum and the xylophone at 7 ft. when he had learnt that he was expected to respond to sound.

This child and his mother were admitted to the Hostel at Ealing (Whetnall, 1954) for a period of training, where the emphasis is on listening rather than watching. With continued intensive efforts by the parents it may be possible for him to acquire speech mainly through hearing, in spite of the lost two years. How much better if this could have happened when the child was 2 months old. The mother should have talked into the child's ear during the period of readiness for listening. A Monopack hearing aid should have been issued very much earlier—at the latest when the child began to crawl (Whetnall, 1955).

The deaf child babbles just as the hearing child but about the time he begins to crawl babbling ceases. This is because the child is no longer close enough to the mother's voice. Babbling occurs in the severely deaf child but in a toneless voice. The absence of babbling is very rare and probably indicates total deafness. The type of aid which should be used in a baby is a Monopack with A.V.C. or some similar device.

May I point out that where, as yet, there are no local facilities, this Hostel is available for any deaf child and the mother from any part of the country, so that no time may be lost.

Our problem is how to ensure that these patients are detected at an early age and start at once to receive adequate training. Knowledge of the way that the normal hearing child learns speech, and of the help that can be given even to a severely deaf child by adequate early auditory training should be known by doctors, nurses and health visitors; teachers of the children must be trained in these methods. Parents will then be on the lookout for any sign of deafness in their child and will seek advice early. (Figs. 5 and 6 show the reactions of a normal and a deaf child to sounds.) The kind of basic knowledge that it is necessary for them to have in order to do this and to co-operate in the training of their child will be briefly described.

The term "auditory training" itself perhaps gives people the impression that it is something that happens in the ear, whereas in fact it is something that happens in the brain, in the auditory cortex and its associated tracts. It is here that memories of sounds associated with their meaning are stored; the use of these memories is called auditory discrimination.

Because a sound is heard it is not necessarily understood but has to be heard over and over again before it becomes so familiar that it can be recognized. This applies to the learning of a foreign language and also the child's learning of its own language. Consider the incessant speech with which a young child is surrounded, often from very close range. The normal child produces the first meaningful words during the period of readiness for speech from 12 to 18 months, but this period has been preceded by a period of "readiness for listening" (Whetnall, 1955) in the first year. At the time that the child is producing his first sounds, which are just vowel sounds, he is already understanding words and sentences.

This stage of facility for learning speech is similar to the stages of development of other functions, manipulating playthings, walking, taking solid food and so on. If the normal first stage is missed there is considerable difficulty in learning this function. After three years the facility for learning speech diminishes, so that by the age of 5 it is difficult to teach a child through its hearing with no previous training and by the age of 7 it is almost impossible.

The importance of this basic physiological principle in the management of the deaf child is the main reason why the otologist must give the lead in this form of training.

The conditions for learning speech through hearing are:

Hearing aid. The child must have a hearing aid and this must be small and easily wearable, for the aid must be worn continually so that he may hear sounds all the time.

Age. It is imperative that the child should have the aid when he begins to crawl, but there is no reason why the aid should not start to be used at 5 or 6 months or even younger.

In these young children, there should be some means by which the aid can be obtained immediately the diagnosis is made. A wait of up to six months sometimes occurs, which obviously loses the time saved by the early diagnosis.

The difference in response by the young and by the older child to the acquisition of a hearing aid is remarkable. The young child likes his aid, wears it all the time and is upset without it. The older child is upset with it and will always be finding excuses for not wearing it. He is past the age at which discrimination can be learnt easily, and all sounds to him are noises. In addition he is a fixed visualizer. The child with an acquired deafness of severe degree will learn to lip-read to help his remaining hearing; but once the congenitally deaf child whose hearing is untrained has been taught only to lip-read, he seems incapable of adding the ability to listen.

FIG. 5.—Reaction of normal child to cup and saucer.

FIG. 6.—Reaction of deaf child to cup and saucer.

It was due to the production of the Medresco hearing aid that so much knowledge is available about the use of hearing aids in children. During the year 1952, 700 hearing aids were issued from our clinic alone to children. Nearly all such children in schools of the London County Council, irrespective of age, received one. Some of the results from this have already been published (Whetnall, 1953) and contrasted with the results of aids issued to younger children at the Audiology Unit.

The child under 2 will keep his babbling voice and speech production and understanding will develop along comparatively normal lines. The child of 2 or 3 who hears the voice at 1 foot or even at the meatus unaided, will make progress to understanding a considerable amount of speech in six to twelve months and this will soon be followed by speech production. The child over 3 has more ground to make up but even so it may be possible to get him ready to go to an ordinary school by the age of 5. Attendance at an ordinary day nursery will be helpful.

In the child over 7 years of age who is severely deaf the aid is of very little value, except as a means of improving the tone of the voice, unless the child can already discriminate between words. Considerable individual training will be required. Children with a less severe loss may not be seen until this age and can be greatly helped. It would be better if they were detected at an earlier age before speech defects develop.

Environment is a major factor in training a deaf child to use his hearing. It is useless to expect a child handicapped by deafness to learn speech under conditions in which a normal hearing child would fail, or would certainly be retarded. In a residential nursery even a child with normal hearing may be retarded in speech, because of the inevitable absence of individual attention.

It is well known that in the absence of special training a child who acquires deafness during the early years of life will lose his speech because he no longer hears his voice or the speech of others. In the same way the congenitally deaf child who by the age of 4 or 5 years has learnt to say a few words may lose his speech if admitted to a school for the deaf. Thus the young deaf child should be admitted to an ordinary nursery school to mix with hearing children.

Size of aid. In 1953, the experimental use of Monopack hearing aids was started by Dr. T. S. Littler and myself. The cases have been carefully followed up and there is no doubt of the success of the experiment. Children like the little aids and even in some of the older children who have been difficult about wearing a larger aid, the results have been gratifying. There is rapid improvement of speech and comprehension of speech because the aid is worn continuously. Children regard them as their lifeline, they do not like to be parted from their aids and some have even been reported as getting into their baths with them on.

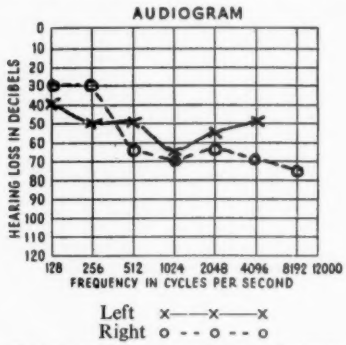


FIG. 7.—I. G. 9 years. Maternal rubella.

Fig. 7 shows the audiogram of a child aged 9 who was deaf following maternal rubella. Many of these children are intolerant of sound and those that have been tested show recruitment. It is essential for these children to have aids with automatic volume control. Indeed it is desirable that all young children should have aids with automatic volume control.

There are *no contra-indications to continual use of a hearing aid.* It has been suggested that care must be taken in using the aid because the hearing may be damaged; no evidence of such damage has been seen among cases treated at the Audiology Unit. We have seen children with a progressive deafness who have never used a hearing aid. There appears to be no reason for recommending short periods of use of the aid except in a few instances. Usually it is better to start right away putting the aid on in the morning and taking it off at night. This saves making the decisions as to when the aids should be on or off. If the aid is a Monopack the child soon comes to regard it as part of his clothes.

Effect of auditory training on tests of hearing.—As the child learns to listen there is an apparent improvement in the child's hearing, not only for speech but for pure tones also (Fig. 8). This change has been described by several other workers (Goldstein, 1939; Wedenberg, 1951; Huizing and Pollack, 1951). It is due to the fact that sounds that have been learnt have become familiar and are now easy sounds and can be understood at greater distances than a new and more difficult sound. The intelligent child will make full use of a few clues.

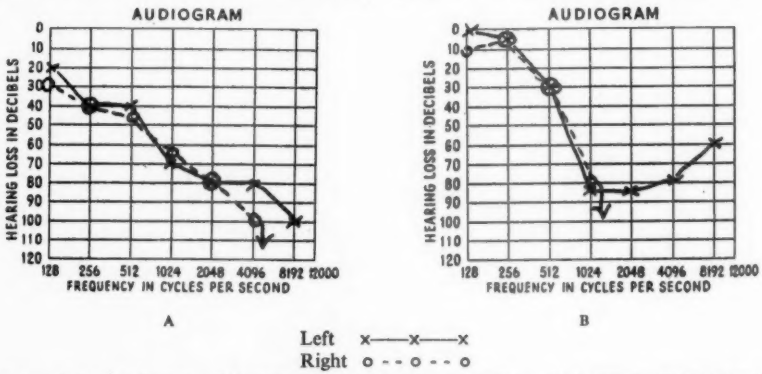


FIG. 8.—J. A. A: 6 years—Discriminates vowels only, no words. B: 16 years—Discrimination within normal limits.

Because of this apparent improvement it is impossible to test a child's hearing accurately until the residual hearing has been trained. Because a child does not respond to speech it must not be concluded that the child never will. There may, in fact, be no response at the

first examination, but all children should be given the opportunity of learning that sounds have meaning, by listening with a hearing aid, for at least a year. In some cases they will be found after several months to have an appreciable amount of hearing.

It is the child's ability to hear and understand speech after adequate training that is important. Many deaf children are never given the opportunity of listening in the way that the normal hearing child does for the simple reason that the first tests of hearing show that the child cannot understand speech. These children are then placed in a school for the deaf surrounded by other children who do not talk, with some individual attention but not the continual individual attention that the child would get at home. Auditory training is doomed to failure if all that the child receives is a few odd half-hours listening practice a week.

Tests of hearing should state clearly whether the tests have been made before or after auditory training.

Usable hearing.—The meaning of this term varies considerably and depends partly on the interpretation of "severely deaf" and partly upon whether the principle of auditory training as just described is accepted.

For example, a child of 2½ years was receiving training by lip-reading only. There was no training of hearing at all; the child had not even got a hearing aid. Yet he responded to an ordinary voice at 1 foot by turning his head and saying the vowel "o". He had been diagnosed as totally deaf. The teacher of lip-reading said, "We never give up hope about their hearing". I think it is more practical to give the child an aid and talk to him. A pure tone audiogram had not been done because, it was said, he had no language.

Pure tone audiograms should not, in any case, be used for assessing the child's ability for understanding speech. There is still a tendency to wait until the pure tone audiogram can be carried out, which loses time; or to look at a pure tone audiogram, which is, perhaps, the first test of hearing the child has ever had, and say how the child must be educated. There are so many other factors to be considered, such as the child's intelligence, personality and environment, that are not shown by a pure tone audiogram, yet which will influence greatly how he will use his hearing. In any case it has been shown that even a pure tone audiogram will improve with training.

For those who are contemplating setting up a clinic here are a few figures from the Audiology Unit to combat the opposition of those who say, "Oh, there aren't many deaf children in this district" (Table I).

TABLE I

Statistical data for 1948 and 1952.

	1948	1952
New cases	57	786
No. of attendances for training and follow up	576	9,247
Hearing aids issued	34	809

The total number of new cases now exceeds 5,000. While a full examination is made of each case, we have had to limit those we can take on to train, but we endeavour to give some training until help elsewhere can be arranged. We always try to take on all children under 5 years of age because we know that as yet there are not many places where they will be trained on these principles. I should like here to pay tribute to the teachers of the deaf who have helped to pioneer this new approach.

Tests of intelligence.—These tests must be carried out by someone who fully understands the communication difficulties of the deaf child and their adverse effect on the child's mental development and personality. Intelligence tests do not measure the intelligence of the deaf child at the first test; for example, a child had an I.Q. of 75 when first tested, and 110 after some years. This illustrates the need for several tests to be made at intervals.

Some cases presenting behaviour problems require repeated testing before even a provisional first result can be obtained. Even then a decision may not be possible and only the response under training can decide the ultimate diagnosis.

The greatest limiting factor in the number of children that can be dealt with is the shortage of staff trained in the method of teaching speech through hearing. These should have had several years teaching normal children. People with nursery school experience would seem to be ideally suitable.

The time seems to be ripe for otologists to express their views about this whole question, and in particular about the need for the training of teachers of the deaf to be adapted to the problems of the deaf child or child with partially impaired hearing, who is being educated in an ordinary school.

These problems must be solved if we are to do our best to help the deaf child to overcome his disability. The child is unable to overcome the disability himself and is entirely dependent on adults for help.

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Mr. I. S. D. Thomson:

In the first nine months of life the normal infant looks at the faces of those around it, but after this age it pays more attention to speech and looks less as time goes on. The profoundly deaf baby continues to study faces till about a year and a half old after which it begins to lose communication with its parents. The habit of dumbness must therefore be averted at the right time for learning to talk, which is the first three years of life.

The profoundly deaf infant who does not start to speak at the normal age is presented for diagnosis early. Less severe deafness may not be suspected till speech defects become apparent at a later age, or even until the increasing tempo of education exposes the defective hearing by forcing the child further and further behind his classmates. Hence, the age at which speech started, and defects in speech, is of diagnostic importance; and a history should also be obtained of what hearing the child has. Help in the differential diagnosis from backwardness is obtained from enquiries about his developmental milestones. The clinical examination should have taken place on another occasion, and so far as possible a hospital atmosphere is to be avoided.

Diagnosis of deafness in infancy is made by comparison with the following standards of normally developing hearing:

Under 9 months old, reflex response to sound gives positive evidence of hearing; and by 6 months old, ability to locate quiet interesting sounds such as voices, bells and pitch pipes is present. Loud sounds are frequently ignored.

By 9 to 15 months, children can automatically locate *quiet* sounds that catch their interest, for example their own name, called quietly, the clink of feeding bottles and musical bells. For some reason the rustle of paper fascinates them. At this age positive evidence of normal hearing can be obtained.

At this stage it is desirable to describe in more detail what I call the "Distraction technique". It was shown to me by the Ewings. The infant sits on its mother's knee, and its attention is lightly held by an assistant offering it simple toys such as bricks. No attempt is made by the assistant to hold the child's attention actively (Fig. 1). When a test sound is made behind and to one or other side of it, or above it, the normal child automatically and quite rapidly looks towards the noise (Fig. 2).

FIG. 1.—The "Distraction technique."

FIG. 2.—Location of quiet sound by an infant with normal hearing.

From 1½ to 2 years, normal children can, in addition, locate unforced consonants at 2 feet distance—"K", "P", "T" and "S"—and can also carry out simple commands such as "Put it on the table" or "Give it to Mummy".

From 2 to 3 years old they respond in words to simple speech spoken quietly at 3 feet.

Until ability to locate sound is present, it is not possible to say that the hearing of a young infant is normal, and because their responses may vary greatly from day to day tests must be repeated if the infant does not come up to the normal standard. As he grows older, interest in loud sound increases but all sounds should be intimate and friendly. Testing for consonants is important as it indicates defects in the upper frequencies of the speech range. Pitch pipes, too, are used for this purpose.

From the age of 2 to 5 simple games of "put and take" with toys are played, the mother taking part at first to show the way. The tester is visible to the child at first and then moves out of his sight. A quiet conversational voice at 3 feet is used first as it is at a level of 50 decibels above threshold, the optimum level for understanding speech (Fig. 3).

As familarity breeds confidence the older children in this group may be introduced to headphones and the game continued with a pure tone signal and an audiogram constructed —though an audiogram, which measures threshold to pure tones, is not an indication of the ability to understand speech (Fig. 4).

FIG. 3.—Tests of hearing by "put and take" games.

FIG. 4.—Pure tone audiometry by games.

Hearing for consonants can be tested by using toys with similar vowel sounds, for example, "horse" and "ball". Testing at any age by repetition of numbers fails, as nearly all numbers have different vowel sounds. However, skilful insertion of "five" and "nine" at the critical distance from the patient helps to overcome this. Amplified speech can be delivered through the audiometer or a hearing aid. The "Peepshow" which produces a binaural pure tone audiogram is suitable for this age group.

When the diagnosis of deafness and, so far as possible, its extent has been made, communication with the infant must be established. The line of communication may have been severed by total deafness, and a makeshift is constructed by lip-reading, if possible before the infant loses visual contact with its mother, or the fragmentary reception is improved by auditory training and artificial aids, to wit hearing aids and lip-reading. Large centres have facilities for the organized parent guidance and auditory training which are so vital to keep alive the tenuous lines of communication and enlarge them until the child takes his place in the nursery school at the age of 3. I am fortunate in having the assistance of the headmistress of the Aberdeen School for the Deaf who shows the parents what can be done for the child and advises them, at a visit to the school.

The infant can be proved deaf by 9 to 12 months old but the degree of deafness cannot be established accurately as yet. Therefore both methods of communication may be used, visual and aural, though for the reasons already given deafness diagnosed at this early age usually infers a profound degree.

First of all the parents are reassured that all is not lost, the connexion between hearing and speech development explained, and the importance of maintaining visual and aural communication emphasized. Stress is also laid on the co-operation of all the family, friends and neighbours, and upon the need for illimitable patience before results appear.

If there is any evidence at all of hearing at testing, such as is evoked by a loud voice close to the ear, with suitable safeguards against breath movement (Fig. 5), the mother is shown

FIG. 5.—Testing with a loud voice close to the meatus.

how to speak clearly to the infant with the face visible but without over-emphasis or mouthing of the words. The words are repeated close to the child's ear and the sense of the words illustrated by actions. Perhaps an illustration to the parents will show the interdependence of the three methods. Firstly I mouth a sentence in silence. Then I repeat it filling in a few vowels which a deaf child may hear because they require a greater intensity of sound than do consonants in their production. Lastly I repeat it with appropriate actions and all becomes clear. It has been calculated that a moderate voice at 6 in. or a loud voice at 18 in. is equivalent to 80 decibels above threshold. Sentences are preferable to single words as the more sound clues given the child the more it can fill in the context, as shown by the 40% critical level in word articulation scores. The same is true for lip-reading.

At this point it is important to emphasize to the parent the difference between demonstration, where sound and lip-reading are underlined by correlated activities, and "signing" which is a silent, private language rendering hearing and lip-reading unnecessary. To show a child a saw and what it is used for, while teaching it the appropriate sound or lip pattern is one thing. To refer to the saw by a silent gesture is another and deplorable matter. Any signs by the child are encouraged as they show a desire to communicate with its parents. The parents should then catch the child's eye and say the word it requires. Similarly any vocal noises by the child, or any lip movements in an attempt at phonation are warmly encouraged and visibly appreciated as the child will associate the parents' pleasure with the kinæsthetic sensation of vocalization. No formal lessons are required at home and the infant is in all respects treated as normal within the above limitations.

As stated, the older a child is when brought for diagnosis, the more hearing it is likely to have, and the more accurately the amount of hearing can be determined. Consequently the assistance from auditory training and hearing aids increases in proportion, and these in turn increase the child's potential for speech, the understanding of speech and the appreciation of sound.

Acquired deafness in the young child, if partial or profound, causes rapid deterioration of speech and dumbness follows within a year unless training is started as soon as possible. The first step can be taken even if the child is still in hospital. Visiting by the parents is of first importance in order that the child, surrounded by familiar voices and faces, can learn to equate the new pattern of fragmented sound with his previous hearing. A few words to the parents along the same lines as those given to the parents of the profoundly deaf infant are a great help.

In conclusion a few words are necessary to put into perspective the future of a child who will end up at school in Grade II. Subdivision into IIA (those who make satisfactory progress in ordinary classes in ordinary schools provided they are given some help ranging from a favourable position in class through hearing aids and auditory training to tuition in lip-reading) and IIB (those who fail to make satisfactory progress in ordinary classes in ordinary school with any or all of these aids) is bound to be finally by performance at school. Whatever method of testing is used in the clinic it cannot take into account the reverberation and resonance present in a classroom nor can it allow for the unselective pick-up by a hearing aid of unwanted noise. I submit that IIB pupils should not only be taught in special classes

but in special classrooms which have been acoustically treated. If not, the fruits of earlier training may be wasted. It may be argued that this is no preparation for the noise and hurly-burly of everyday life but I have yet to hear of a new school for ordinary children being built next to a riveter's yard. We as a specialty should make it a professional matter to press for increased facilities both for teachers of the deaf to assist all Grade II children and for suitable educational environment for Grade IIB. Generally speaking only Grade III children are adequately catered for educationally and I fear the criterion for proper educational facilities for the handicapped child is its helplessness. Surely a Grade II child will make a better, more useful citizen than, for instance, a mentally defective child.

Dr. D. C. Kendall:

The field of audiology is perhaps unique in that it includes workers from a relatively large number of different professional disciplines. Otologists, physicists, electrical engineers, educationists and psychologists have all been required to contribute something to our understanding of deafness and the effects of deafness.

Essentially, the part of the psychologist is to discover, identify or call attention to some of the human processes and problems in the measurement of hearing loss and educational treatment of deafness. He is likely to leave their solution to his colleagues in the medical or educational fields.

In recent years two significant trends of interest and research stand out in the work that has been done on the educational treatment of deafness. First the increasing emphasis on the early diagnosis and training of the congenitally deaf child, and second the increasing use of residual hearing in training programmes with all ages of deaf children, but particularly during the pre-school years. The normal infant has already begun to comprehend speech by the age of 12 months: by the age of 5 practically all the basic speech patterns have been established, as well as a sizable vocabulary. Speech and communication cannot be separated from mental—by which I mean intellectual, social and emotional—development. If speech begins as a response to particular environmental conditions, it rapidly becomes a stimulus to growth in almost all fields of development. The handicap of deafness, in that it inhibits communication, and particularly the development of speech and language, strikes at the root of intellectual and social development. Clearly the first aim of educational treatment is to stimulate speech development by all available means, and to begin this process as early as possible and here I would like to support Miss Whetnall's insistence that auditory stimulation and training be given during the early months, for this is precisely the stage when auditory experience should contribute to mental development as a whole, and the development of speech and language in particular.

The early treatment of deafness depends primarily upon reliable diagnosis of hearing loss. Broadly speaking, we can distinguish three main types of hearing tests which have been used, tests which involve the observation of response in a "free" situation to incidental, distracting sound stimuli, tests which are designed to produce threshold or near-threshold responses to pure tone and other stimuli through a modification of audiometric techniques, and tests designed to measure hearing for speech.

In order to understand the difficulties of audiometry with young children, we must first appreciate the simple learning situation contained within the pure tone or speech test with an adult. Here a motor or verbal response is tied to an auditory stimulus: the tie-up between the two is explained verbally to the subject and requires a conscious act of selection or judgment on his part. In other words there are two processes, communication and judgment, which take place at a relatively highly-developed level of learning, and which yield the basic data from which response levels or hearing loss curves can be deduced. However carefully the instrument which produces the stimulus is calibrated, however rigidly the conditions under which the signal reaches the ear are determined, we are still left with a measurement whose validity rests upon subjective judgment on the one hand and the particular psychophysical method used to obtain this judgment on the other. And it is this point which leads to the greatest difficulty in obtaining responses, let alone threshold responses, in children with whom communication can only be established with difficulty and at a primitive level, and whose thinking is tied to the concrete perceptual world, incapable of abstraction. The first essential is to devise test techniques which are related to the developmental level of the children with whom we are concerned.

In the normal infant response to sound shows a characteristic development during the first 9–12 months (Gesell *et al.*, 1941; Buehler and Hetzer, 1935). Leaving on one side individual differences in "responsivity", we can distinguish between reflex responses to loud stimuli (which show an increasing definition of response with age) and a gradual process of perceptual discrimination, consisting on the one hand of a development of interest in "meaningful" sound stimuli—speech, musical sounds, sounds associated with other ex-

periences such as feeding, and on the other hand of a process of habituation—a rejection of response, so to speak, to unimportant, meaningless background noises.

In testing hearing, certain crude and approximate indications of hearing status may be reached by comparing the responses of a particular child suspected of deafness with the responses expected of a normal child to the same stimuli. In the clinic, in suitable acoustic conditions, the sound stimuli can be roughly defined in terms of loudness and pitch, and these tests, when carried out by experienced workers, are of the greatest value in ascertaining deafness in early infancy. It is most important to make a careful assessment of the whole developmental status of the child before evaluating such test results since there are many factors—emotional disturbance, intellectual retardation, environmental deprivation which may affect response to sound.

The second type of test technique, that which involves a conditioning or learning situation, either at an autonomic level, as in the psycho-galvanic skin resistance test developed by Bordley and Hardy (1949) or at a higher level as in the "Peepshow" test of Hallpike and Dix (1947) or the activity test developed at Manchester (Ewing and Ewing, 1947; Kendall, 1952), has been adequately reported in the literature. The P.G.S.R. technique has been used successfully with children well below the age of 3: the other learning tests cannot usually be attempted much below the mental age of 3 but various surveys (including an unpublished one from Manchester) have shown that reliable pure tone audiograms can be obtained with children of this age and upwards. Again, one cannot overstress the importance of developmental assessment in interpreting results, nor the necessity of using workers experienced and skilled with young children.

Thirdly, in the battery of available tests there is the use of some form of speech test, particularly, of course, with partially deaf children. Live voice, or monitored live voice, has often been used incidentally in diagnosing deafness in young children, but it is only relatively recently that attempts have been made to construct word lists, administered under more strictly controlled conditions. In a pilot experiment at Manchester we found an encouraging response to a simple test situation involving the selection of one of a group of objects, with the speech signal recorded on tape and reproduced by loud speaker via a calibrated attenuator. Three monosyllabic word lists were prepared and thresholds established for normal children of $2\frac{1}{2}$ years and upwards. It would appear that this technique has some value as an alternative screening test, and, in addition, can be used to record objectively the results of auditory training.

Audiometric tests with young children yield, then, tolerably reliable estimates of hearing loss which can be used diagnostically and as a basis for training. Since the term "deaf" refers to a widely ranging variable condition extending from relatively insignificant hearing loss to total loss of hearing, with a variable age of onset, it is convenient to adopt some sort of classification system when making general statements about any particular deaf population. Such systems are arbitrary and are nearly always unsatisfactory when used prognostically or as if they were absolute. They would seem, however, to have some value when used in a limited context as, for example, when categories of hearing loss are related to the audibility of speech in normal conversational conditions, or when a category of hearing loss suggests the most appropriate educational or training programme. The terms "deaf", "partially deaf", "hard of hearing", "severely deaf", "profoundly deaf", "totally deaf", are all to be found in technical as well as popular literature, and they are often left undefined. In terms of his handicap and his educational needs I think that there is some sense in distinguishing between the child who, unaided, hears and can recognize many of the speech sounds in his normal environment: the child who hears loud sounds only; and the child whose hearing response is confined to sounds of intensities so great that they are rarely found in normal experience. Yet to-day all these children may receive the same label, and may be found in the same school or even the same class. In our research into the mental development of young deaf children at Manchester (Kendall, 1953) we used a simple, coarse classification on the basis of the average of the threshold responses to pure tones or pitches of 500, 1,000 and 2,000 cycles per second. This gave five categories:

	Db	
I. Average loss significant but less than	30	} Partially deaf
II. Average loss	30 – 60	
III. Average loss	60 – 90	Severely deaf
IV. Average loss	90	Profoundly deaf
V. No response to sound.		Totally deaf

Some of these categories (e.g. IV and V) could readily be combined for certain comparative purposes. Although this grouping is arbitrary it is related approximately to the stage and type of language development to be expected from children falling into one of these categories, and receiving normal environmental stimulation rather than special training. Thus children

in category I would normally develop speech that was not defective, and hearing difficulty would only show up in poor acoustic conditions. Children in category II would tend to develop defective or grossly defective speech, and would only comprehend speech in optimum listening conditions. The children in category III, who heard only the louder speech sounds, would be unlikely to recognize words or develop speech without appropriate training. They live, however, in a world of sound, and their own vocal utterance often reflects the kind of stimulation they receive. Finally the children in categories IV and V live in a largely silent world (particularly as far as speech sounds are concerned): typically in normal home conditions where auditory training is not given they do not develop speech or comprehend speech except through lip-reading, and typically also the normal vocalization patterns of early infancy either die away or persist in forms that are unmodulated and increasingly without intonation.

This is a rough and ready classification, resting upon hearing status. The implications for speech development are naturally revolutionized as soon as special training, especially auditory training, is given in the pre-school years.

I would like now to refer briefly to some of the results of the work on mental development of young deaf children carried out at Manchester between 1948 and 1953 (Kendall, 1953). The main purpose of this investigation was to carry out a comprehensive survey of the intellectual, social, emotional, linguistic and motor development of deaf children in the age range of 18–65 months. (Although some partially deaf children were included in the survey, I shall confine myself to the results with children in categories II, III, IV and V.) The investigation consisted of two parts, (1) the administration of performance tests of intelligence or what Gesell calls adaptive behaviour to large groups of deaf and normal children, and (2) a more detailed study of the general development of 180 deaf children and a group of normal children matched for age and socio-economic status. The deaf population was drawn from two sources, (1) referrals to our clinic and (2) children attending nursery schools for the deaf in Manchester, Berkshire and London. The control group consisted of (1) Manchester children, (2) neighbours of the deaf children, and (3) children attending day nurseries in Salford.

Summarizing under the heading of intellectual development, two performance tests, a modification of the Merrill-Palmer (Stutsman, 1931), and the Atkins (1931) test, were found to be suitable for deaf children, and were standardized with pantomime directions. Excepting a group of children who were so retarded that they were unable to attain any score on the tests, there was no significant difference between the mean scores of deaf children and those of normal children at any age level, nor of mean I.Q.s. There was, however, a significantly high proportion of deaf children who were seriously mentally retarded or whose development showed gross abnormality (5·6% compared with 1·2%). One hypothesis to account for this would be that in certain cases the failure of the brain to develop is directly related to deafness perhaps of central origin. There was no significant relation between hearing loss and intelligence, as measured by the tests used.

These results are in general agreement with the work of other English psychologists, Hood (1949), Murphy (1952) and Gaskill (1952), working with older children.

But while it is important to realize that deaf children do as well as hearing children on performance tests of simple sensorimotor skills involving the perception of size, colour and shape relationships, fitting, manipulation, dexterity and problem solving, this should not obscure the fact that their intellectual status (as opposed to capacity) is not, by and large, equivalent to that of normal children. Our survey showed that the greatest differences between deaf and hearing children were, not unnaturally, found in the area of speech and language development. Not only does speech as communication tend to be grossly retarded, but also affected are the many processes of abstraction, relational thinking, conceptualization and imagination which are facilitated and refined by the use of language. The young deaf child is tied far more closely to the here and now of his immediate perceptual experience than is his normally hearing contemporary, and his comparative failure to develop the referential framework of language carries inevitable consequences for his mental development as a whole. However, the most serious effect of linguistic retardation at the pre-school level is, perhaps, in the area of social and emotional development.

As Lewis (1936) has shown the important pre-verbal stages of speech are intimately bound up with the expression and satisfaction of physical needs, while the later "naming" stage reflects the child's growing ability to differentiate things in his perceptual environment, and his awareness of the part played by other people in shaping this environment. In these early years speech is at the same time the instrument used by the child to express his needs which depend upon others for their satisfaction, and to make explicit his differentiation of himself from his social environment by means of defiance or objection. The overt signs of maladjustment in stress situations—tears, temper tantrums, physical aggression—are gradually canalized into verbal channels, until the epithet or term of abuse is—almost—as

satisfying to the child as the direct physical assault. A child becomes aware of the existence, the reactions, the feelings and the personalities of other people through his direct experience of their behaviour in relation to his own behaviour and of their interpretaton or comments upon events. It would be difficult to overemphasize the part played by speech in directing or modifying the course of early social development, and hence of laying the foundations of a healthy emotional development. From the latter half of the first year onwards the hearing child learns to accept speech as an integral part of his experience, and to use it to lay bare his deepest feelings. Although the first stages of the comprehension of meaning must necessarily occur in a direct intimate relationship between the child and his parents, later developments include the assimilation of words incidentally—picked up, as it were, out of the flux of daily life.

For the deaf child, however, communication is restricted to an extreme degree, even during the earliest stages. In the group of severely and profoundly deaf children studied no child had attained by 5 years of age the standard of comprehension or use of speech reached by the average hearing child of 2. Such skill in verbal communication as was achieved was the result of patient and painstaking training on the part of the parents, few of whom enjoyed the benefit of help in the home or in the management of other children in the family. For the child gestures were the primary means open to him for making his wants known to other people—a means of communication which nearly always depends upon a common perceptual environment for the two people involved, and which is capable of misinterpretation but incapable of expressing the finer distinctions between objects, feelings or intentions. Gesture inevitably anchors its users to the plane of the immediate and the concrete; it cannot easily convey the future, the past or the hypothetical. It can replace some of the early functions of speech for the child, but it cannot of its very nature open the door to the mature sophisticated social adjustment of the hearing child, able to respond quickly to new situations or changes in climate of opinion.

It is evident that there are serious restrictions upon the deaf child's communication with other people in his environment and upon his ability to understand what other people wish to convey to him. This has the effect of limiting his relationships with other people and his opportunities of learning from these relationships. If we add to these the external restrictions imposed because of his handicap (which prevent him from playing outside his home with other children in the street or park), and tend to throw him into a close dependent relationship with his mother, we see that it is hardly surprising that there is a higher proportion of maladjustment among the deaf than among the hearing during the pre-school period. His desires and feelings can find only incomplete expression through gesture, vocalization or rudimentary speech. He is likely to misunderstand or misinterpret the intentions of other people; in times of stress it is frequently difficult for them to make reassurance sufficiently specific. He lacks the equipment of the hearing child to deal with the novel, unfamiliar or unstructured situation; in such situations he is dependent to a considerable extent upon others for his successful adjustment. He tends to have few opportunities for contact with other children outside his immediate family circle, and little chance to participate in co-operative group play. If his deafness is adventitious it is probable that he has spent a period in hospital, separated from his parents in addition to the possibly traumatic experience of sudden loss of hearing. Failure to recognize his handicap quickly may lead to misunderstanding on the part of his parents and others, and to inappropriate treatment of his difficulties.

These points were clearly brought out in the survey. Social-emotional development was studied partly by means of developmental schedules similar to those of Gesell, and partly by a schedule of emotional symptoms and behaviour problems modified from those devised by Cummings (1946) and Blatz and Griffin (1936). The records showed a consistently higher incidence of behaviour problems and symptoms of emotional disturbance among the deaf children than among the hearing children, and one moreover which did not tend to decline with age in the deaf group as it did in the hearing group. This result is consistent with that obtained by Murphy working with deaf children of primary school age, and does, I think, underline the importance of including the mental health of the deaf child in any consideration of his educational treatment.

I believe that the first essential is to recognize that the range of these problems that confront us in work with the young deaf child is large and complicated by factors such as hearing loss, age of onset of deafness, and limitation of experience. They can only be overcome by an extreme flexibility of approach, and by relating the handicap of each child to his own unique development. If language is to be an integral part of mental development, the deaf child's experience of speech must begin from the earliest months. Residual hearing should be developed through auditory training and used side by side with the rest of perceptual experience, and not be delayed until the classroom or the group hearing aid. Perhaps too dogmatically, I feel that the right place for the deaf or partially deaf child during the pre-

school years is at home and with his parents and not in a residential school. This implies throwing the onus of his training on to the parents, and hence the provision of more and more clinics where they can receive guidance, help and teaching. Theirs is a great burden, and one that has too often in the past received scant recognition. I cannot believe that the rehabilitation of any deaf child can fully be completed without their co-operation.

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Dr. I. Simson Hall (Edinburgh) said that one of the problems was the finding of these children. Miss Whetnall had said that if a clinic was opened the children would come but in their experience they found they were getting children much older so that development of speech was carried on under a severe handicap.

He agreed with Mr. Thomson that the otologist was frequently the last to see the child and it seemed to Dr. Hall that the teaching of simple screening techniques to those who first handled the child, namely the nurses and health visitors, was the best way to ensure early recognition of deafness.

There was no doubt that in the case of the severely deafened child the problem of teaching was to a great extent the teaching of the parents, and again it seemed that those responsible for the children had to make up their minds whether this was to be considered a medical or an educational problem. He felt it was predominantly educational and was best carried out in conjunction with schools for the deaf.

Dr. C. S. Hallpike sympathized with the parents of a young child with defective speech, thought to be due to deafness, who were anxious that something should be done without delay.

The problem was best regarded as a tactical one: how best to use a limited supply of expert personnel for the greatest good of deaf children as a whole. The experience of his colleague Miss Dix and himself was, in the main, concerned with children over the age of 3 in whom it was nearly always possible to carry out pure tone audiometric tests by means of the "Peepshow" procedure.

In the very young children, who were Miss Whetnall's particular concern, the difficulties were very much greater. These children, being so much younger, required much more individual attention and since accurate hearing tests were not possible, the application of hearing aids was carried out at a considerable disadvantage with inevitable loss of efficiency.

It was, therefore, necessary to ask whether educational efforts made at this stage would not be more effective if deferred for a year or so until, at any rate, a reliable pure tone audiogram had been obtained. The answer to this question depended upon what was thought of Miss Whetnall's central thesis. If he had understood this correctly, the capacity for normal speech depended upon certain vital developments in the central nervous system. These developments were dependent upon normal hearing and took place in the first three years of life *and at no other time*. From this it followed that a young child with a hearing defect, uncorrected by means of a hearing aid, would suffer during this critical period a developmental defect which was irreparable. If they accepted this then it was plain that what was lost in this period would be lost for ever and that their efforts to prevent this must not be delayed. If, on the other hand, this was not quite the true picture and the loss sustained during this period could be later regained, then the same urgency did not exist and results as good might, in the long run, be obtained if educational treatment was deferred for a limited period, i.e. until the age of 3 or 3½.

He did not deny that a partially deaf child intensively treated from the age of 2 or less would at first appear to establish an advantage over a comparable child in whom similar treatment was delayed until the age of 3. This, however, was not the point. What was needed was a comparison between these children at the ages of 10 years or so. If then no significant difference could be demonstrated in their speech and other attainments, it would be necessary to conclude that the extra attention received by the one child before the age of 3 had been unnecessary.

No doubt Miss Whetnall and her colleagues had this point in mind and the scientific basis of their thesis would in due course be established by means of adequate statistical studies.

In the case of their own practice they were sometimes asked to see children under the age of 3 in whom accurate hearing tests were impossible. In such cases they generally explained to the parents that no definite opinion could be given upon the state of their hearing and its relationship to their speech and other defects. They asked to see the child soon after it had attained the age of 3 and then expected to be able to obtain a reliable estimate of its hearing capacity. In the meanwhile they generally advised the parents that the possibility, or even the probability of a hearing defect had to be accepted. They advised them further, that until the child had reached the age of 3 years or more, they should treat it as a case of partial deafness, i.e. the parents should keep it with them, play with it as much as possible and speak to it clearly at a close range, being sure always that the child could see their faces. Their words should be simple, clearly articulated, and accompanied by suitable gestures.

In this way, Dr. Hallpike felt that the child's progress would not be significantly delayed pending the further programme of education based upon adequate audiometric tests which were usually possible between the ages of 3 and 4 years.

When the parents were intelligent, they believed that this advice was sound; in other cases, however, there seemed no doubt that parental education was essential and in this respect the training centres suggested by Miss Whetnall should be of very great value.

When they saw such children again, soon after the age of 3, audiometry generally yielded one of two results:

(1) *Normal hearing*, in which case their advice was that deafness was a negligible factor in their backwardness in speech.

(2) *Defective hearing*. In this event, depending upon the degree of deafness, they advised the education authorities that arrangements should be made for the child's education in a school either for partially or severely deaf children.

The responsibilities of the education authorities were not light ones, and it might be that they were justified in feeling that before undertaking them they should be provided with a reliable measure of the child's deafness.

The President said that the otologist would have to take a leading part in the solution of this problem if satisfactory results were to be obtained. This would involve working in close collaboration with the education authorities, public health authorities, welfare workers and to a lesser extent the regional boards and if a liaison committee of the otologists and the various authorities could be formed in each region much would be achieved. Perhaps one of the greatest difficulties they would meet would be to ensure an adequate supply of trained teachers of the deaf.

[*February* 3, 1956]

A DISCUSSION was held on **Chronic Adhesive Otitis Media.** Papers were read by Mr. I. P. J. MACNAUGHTAN, Mr. TERENCE CAWTHORNE, and Mr. C. S. HALLPIKE and Mr. M. SPENCER HARRISON. These papers will be published in the *Journal of Laryngology and Otology*.

The following took part in the discussion: Mr. S. MAWSON, Mr. I. SIMSON HALL, Mr. I. B. THORBURN, Mr. C. A. HUTCHINSON, Mr. R. F. J. MARTIN, Mr. H. V. FORSTER, Professor F. C. ORMEROD and the PRESIDENT. Mr. MACNAUGHTAN and Mr. CAWTHORNE replied.

[*March* 2, 1956]

A DISCUSSION was held on **Conservative Surgery in the Management of Ménière's Disease.** The opening papers, read by Mr. ROLAND LEWIS, Mr. M. SPENCER HARRISON, Miss M. R. DIX and Mr. TERENCE CAWTHORNE, will be published in the *Journal of Laryngology and Otology*.

The following took part in the discussion: Mr. R. L. FLETT, Mr. M. ELLIS, Mr. W. C. GLEDHILL, Mr. P. H. GOLDING-WOOD and Mr. T. J. WILMOT; the opening speakers replied.

Section of Proctology

President—HAROLD DODD, Ch.M., F.R.C.S.

[*January* 11, 1956]

Carcinoma of the Anorectal Region Arising from the Intramuscular and Apocrine Glands (Two Cases).—HENRY R. THOMPSON, F.R.C.S.

The anal intramuscular glands are simple tubular, sometimes branched glands, opening into the anal canal at the crypts of Morgagni, or below this. They are lined by cuboidal or transitional epithelium and may penetrate both internal and external sphincter muscles. They were first described by Herrmann and Desfosses in 1880. Johnson (1914) gave a full description. The significant ætiological association with perirectal suppuration and fistula-in-ano has been recognized in outstanding papers by Sir Charles Gordon-Watson and Harold Dodd (1935) in this country, and by Tucker and Hellwig (1935) and Hill, Shryock and ReBell (1943) in America. The possibility of carcinoma arising in these glandular structures is also recognized and Scarborough (1941) and Willis (1948) both described such tumours.

The apocrine glands which surround the anus are modified sebaceous glands, their secretion being derived from disintegration of portions of the epithelial cells lining their ducts and acini. These glands are vestigial remnants of the scent glands associated with sexual attraction in some animals: the thick secretion of the perianal glands certainly has a very characteristic odour. The apocrine glands are situated deeply in the corium of the perianal skin. The distal ends of their ducts may open into a hair follicle or empty on the free surface of the skin. Benign tumours of the apocrine glands have been reported, usually under the term benign adenoma or hidradenoma, but I have not been able to find any record of a carcinoma derived from the epithelium of an apocrine gland.

The two cases shown presented as atypical forms of anorectal carcinoma. They had, however, certain clinical features which, it is believed, are characteristic of carcinoma arising in an anal intramuscular or an apocrine gland. The recognition of such a pathological possibility, together with distinctive clinical signs, simplifies what might otherwise be a puzzling diagnostic problem.

The following similar clinical features were observed in the two cases:

(1) Six months' symptoms of gradually increasing anal pain, closely resembling that of a fissure-in-ano.

(2) A shallow atypical ulcer at the anal margin, resembling a fissure-in-ano, but with differences in position, size and shape (Fig. 1).

(3) Extensive induration of the perianal tissues suggesting the differential diagnosis of a chronic granuloma such as lymphogranuloma, syphilis, actinomycosis, tuberculosis or Crohn's disease.

(4) Cords of palpable growth extending up in the scroto-crural sulcus to enlarged glands in the groin.

(5) Obtaining material for a biopsy was difficult and could only be achieved under anæsthesia; in one case no malignant tissue was found at the first biopsy and this delayed diagnosis and treatment.

The expected histology of malignant ulceration at the anal margin is one of squamous or of basal cell carcinoma. If the pathologist's report, however, is that of adenocarcinoma and downward growth from a rectal cancer can be excluded, the possibility of the growth arising from a sweat gland, apocrine gland or anal intramuscular gland should be borne in mind. Chronic symptoms of gradually increasing anal pain associated with an extra-rectal lump or perianorectal induration should suggest the diagnosis; or, put another way, an anorectal lesion suspicious of malignancy but with none of the well-recognized clinical features and giving rise to difficulty in obtaining biopsy material, should suggest the diagnosis of a carcinoma arising in an anal intramuscular or apocrine gland.

It is exceptional to find clinical evidence of direct extension of growth between a squamous carcinoma at the anal margin and secondarily involved superficial inguinal lymphatic glands and if there is clinical evidence of such direct extension between a primary anal tumour and secondarily involved inguinal glands, as in the two cases described, then this may prove to be another clinical sign suggestive of the condition.

CASE REPORTS

Case I.—E. M., aged 52. Docker.

Six months' history of pain, at first on defæcation, but becoming progressively severe and continuous and interfering with sleep. Examination showed a ragged ulcer at the left

margin of the anus with considerable induration around the left anterior anal canal and left buttock. There were enlarged hard lymph nodes in both groins and whipcord-like induration extending upwards in the subcutaneous tissue from the anal margin to the left inguinal nodes.

Operation (26.10.55).—Abdomino-perineal excision and bilateral superficial inguinal lymph-node dissection in continuity, with flush aortic ligation of inferior mesenteric artery, removal of pre-aortic, internal iliac and obturator lymph nodes. Suprapubic cystotomy was done at the time of operation.

Post-operative complications.—Urinary retention and infection, œdema of penis and scrotum, sloughing of perineal wound, infection and second intention healing of groin wounds.

Pathological report.—The only visible surface lesion was a small ulcer half an inch in diameter on the anterior anal margin. Longitudinal section through the anal canal and rectum showed a white tumour 2½ in. in length situated in the internal sphincter and extending upwards in the circular muscle coat of the rectum and downward into the perianal tissues (Fig. 2). Secondary deposits were found in three hæmorrhoidal lymph nodes, in two of the left inguinal nodes, and in one of the right inguinal nodes.

Histology.—The tumour varied in its histology in different regions but consisted at its site of origin of cuboidal or polygonal cells arranged in a tubular pattern so that the growth could be identified as an *adenocarcinoma*, though not resembling any of the usual types of rectal cancer. At a higher level, where it was spreading within the sphincter muscles, it consisted of more pleomorphic cells showing no definite glandular pattern, many of them vacuolated as if undergoing degeneration (Fig. 3).

In his report, Dr. Cuthbert Dukes said that this was a very unusual tumour. Judging from the dissection it appeared to have arisen within the internal sphincter muscle and spread both downward and upward beneath intact mucosa and epidermis. The ulceration at the lower end of the anal canal was partly traumatic in origin and probably related to the previous biopsy. If, as seemed most likely, the primary site of origin was within the internal sphincter muscle, then the growth should be regarded as a primary carcinoma arising from intramuscular glands. The cytology of the tumour is in keeping with this site of origin.

Present condition.—An ill-looking man with a malignant left pleural effusion. The paramedian, both inguinal and perineal scars are well healed; there is almost certainly a small recurrence in the left inguinal region.

Added note.—He subsequently died on 3.2.56, three and a half months after operation and nine and a half months after onset of symptoms, with secondaries in the lungs and liver. In spite of a pre-aortic clearance at the time of his operation, there were small deposits in three macroscopically uninvolved pre-aortic lymph nodes.

Case II.—J. P., aged 43. Dustman.

Six months' history of gradually increasing anal pain aggravated by defæcation. Eleven weeks after onset of symptoms, presented at hospital with a shallow ulcer at the right lateral anal margin, with an unusual degree of induration (Fig. 1). A negative biopsy at that time allayed the suspicion of carcinoma. Increasingly severe symptoms and extension of the induration led to further consultation and a second biopsy.

In his report on this second biopsy, Dr. Cuthbert Dukes said that the sections showed the presence of an adenocarcinoma apparently arising from some glandular structure attached to the skin. The growth had a tubular pattern consisting of cuboidal cells with hyperchromatic nuclei, mostly arranged as a single lining to the tubular spaces. In some regions the lining epithelial cells appeared to be breaking down and discharging part of their substance into the lumen as occurs in apocrine glands. This was a very unusual type of tumour and the most likely pathological diagnosis was either carcinoma of the sweat glands or apocrine glands, probably the latter (Fig. 4).

At the time of diagnosis, the lesion presented as a shallow ulcer at the right lateral anal margin with a marginal zone of nodular, hypertrophic-looking skin. There was considerable induration extending round the front half of the anus and out into the right buttock with a nodular cord of tissue extending upwards along the right side of the scrotal raphe in the scroto-crural sulcus to the inguinal region.

The patient was referred to Sir Stanford Cade at the Westminster Hospital for consideration of radiotherapy.

Summary of treatment.—To the anal region, maximum tumour dose 7,550 r with the 2 million volt Van de Graaff generator. To the groins, maximum tumour dose 2,790 r to each groin with 30 curies telecobalt unit, the overall treatment time being sixty-two days.

Present condition (almost three months after onset of treatment).—A healthy-looking man

FIG. 1 (*Case II*).—Perineum in lithotomy position showing anus with ulceration on right side and cord of tissue extending up in right scroto-crural groove.

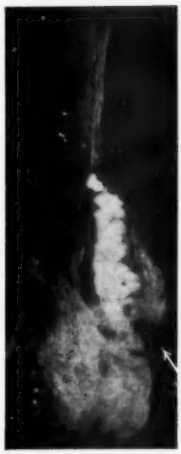

FIG. 2 (*Case I*).—Longitudinal section through anal canal and rectum showing white tumour replacing the internal sphincter muscle and extending upwards in the circular coat of the rectum and downwards in the perineal tissues.

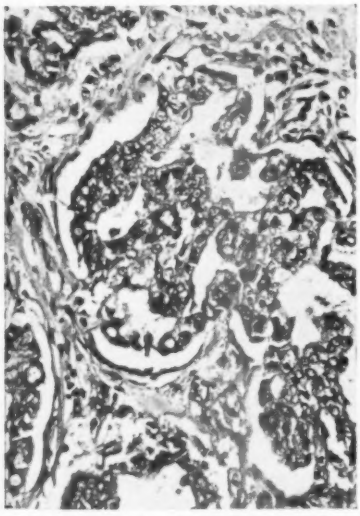

FIG. 3 (*Case I*).—Carcinoma of anal intramuscular gland. ×180.

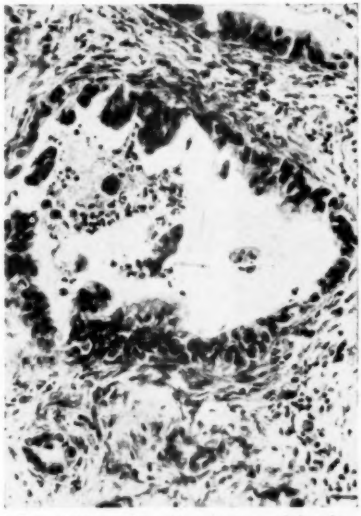

FIG. 4 (*Case II*).—Carcinoma arising in apocrine gland. ×180.

with almost complete regression of the primary anal lesion, no enlarged inguinal lymph nodes, but still a cord-like residuum of growth extending up from the base of the right side of the scrotum towards the right groin. He has, however, a considerable amount of discomfort which makes the sitting position painful.

Added note.—This residuum of growth has been treated with the 30 curies telecobalt unit to a maximum dose of 4,723 r over a treatment time of thirty-one days.

Comment.—The prognosis is probably unfavourable in all cases of carcinoma of the intramuscular glands or apocrine glands. In both the reported cases there was a delay in diagnosis:

(1) from the failure of the patient to report insidious symptoms,

(2) from delay in diagnosing the condition at consultation, and

(3) from the difficulty in obtaining biopsy material.

Similar delay is likely to arise in other cases because a surgeon is naturally reluctant to carry out a radical excision leaving the patient with a permanent colostomy unless there is definite evidence of malignancy or of some other disease with no possibility of natural healing. Even with earlier surgical treatment the prognosis is likely to be bad because of rapid spread which appears to be a characteristic feature of carcinoma derived from the glands opening into the anal canal.

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Carcinoid of the Rectum with Lymphatic and Liver Metastases.—W. B. GABRIEL, F.R.C.S., and B. C. MORSON, D.M.

History.—L. A., male aged 48; two years previously had attended St. Mark's Hospital for treatment of rectal bleeding; this was considered to have been due to hæmorrhoids which were injected. Sigmoidoscopy at that time (September—November 1953) was negative to 6 in.

In September 1955, he returned with a history of recurrent bleeding at defæcation and a prolapsing pile.

On examination.—Moderately advanced right anterior hæmorrhoid with a related skin tag. Sigmoidoscopy was negative at the first attempt, but at the next visit a small friable and ulcerated neoplasm with raised margins was seen anteriorly at 5 in. from the anus.

Biopsy.—Malignant tumour of the carcinoid type. The argentaffin and diazo reactions reveal the specific granules of a carcinoid tumour.

Urine test for 5-hydroxy-indole-acetic acid (5-H.I.A.A.).—Normal.

Operation (13.10.55).—Laparotomy revealed two secondary deposits in the right lobe of the liver, the larger being a pearly-white umbilicated mass 5 cm. in diameter just above the gall-bladder. The primary growth was at the peritoneal reflection; it was mobile and about $1\frac{1}{2}$ in. in diameter, with overlying peritoneum adherent and puckered. A perineo-abdominal excision in one stage was carried out. Before closing the abdomen the appendix was

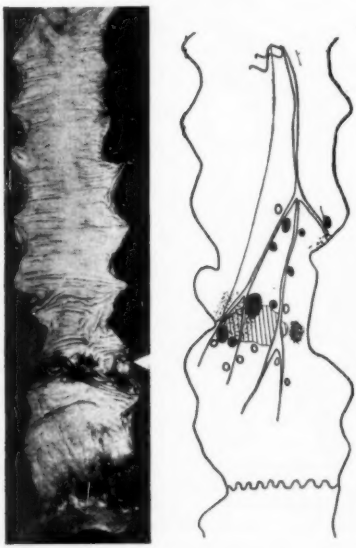

FIG. 1.—Showing the deeply ulcerated carcinoid tumour at the level of the peritoneal reflection and (on right) the lymph node dissection; 9 lymph nodes contained metastases—C2 case.

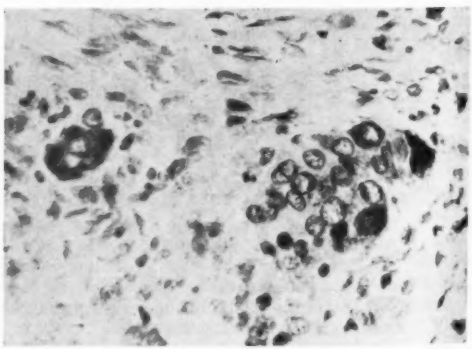

FIG. 2.—Clumps of tumour cells, some of which contain cytoplasmic granules typical of carcinoid. Diazo method. ×457.

removed (clinically it appeared to be normal) and a small fragment for biopsy was removed from the larger of the liver secondaries.

Pathology (Fig. 1 R. and L.).—Deeply ulcerating tumour in the recto-sigmoid, 1 in. in diameter, involving the anterior and lateral aspects. Metastases in 9 lymph nodes (C2 case).

Histology (Fig. 2).—Sections show a tumour composed of small cells, with a tendency to tubule formation. There is variation in size and shape of the nuclei, and mitoses are found easily. Cytoplasmic granules, typical of carcinoid of the appendix or ileum are seen but not in every cell. There are large areas in this tumour in which no cytoplasmic granules can be seen. These granules have been demonstrated by the diazo and silver impregnation methods. Biopsy of the liver shows tumour cells similar to those in the primary tumour.

Subsequent progress.—The patient made a good recovery from the operation and to date there is no appreciable increase in the enlargement of the liver. Urine tests since operation show the level of 5-H.I.A.A. within normal limits.

Comment.—In their behaviour rectal carcinoids may be benign or malignant. The small nodule in the rectum removed by local excision and subsequently reported by a pathologist as a "carcinoid" or "atypical carcinoid of rectal type" seldom seems to recur. About 20 such cases have been followed up at St. Mark's hospital. These small tumours have histological features which are atypical in comparison with carcinoid tumours of the small intestine and appendix. Firstly, in the majority of cases of rectal carcinoid it is not possible to demonstrate the typical cytoplasmic granules. Secondly, the tumour cells form a different pattern, with a greater tendency to tubule formation among other features. The histology of these tumours suggests a relatively benign lesion.

The example of rectal carcinoid described here shows the histological appearances of a malignant tumour. It is also of particular interest because the diagnosis of malignant carcinoid of the rectum was made on material removed at biopsy and this led to a radical operation.

The granules seen in histological preparations of carcinoid tumours are probably an artefact produced by the inter-action of formalin and a substance called 5-hydroxytryptamine. Recent reports suggest that patients with metastasizing carcinoid tumours secrete excessive amounts of 5-hydroxytryptamine into the blood and that this may cause humoral manifestations such as flushing, patchy cyanosis, diarrhœa, and in the later stages, pulmonary stenosis. The 5-hydroxytryptamine circulating in the blood of patients with metastasizing carcinoid tumours appears in the urine as 5-hydroxy-indole-acetic acid or 5-H.I.A.A. It has been suggested that urine tests for this substance may be of value in the diagnosis of carcinoid and in the detection of post-operative recurrence.

This case of carcinoid of the rectum has none of the humoral manifestations of metastasizing carcinoid. Urine tests both before and since operation have shown consistently normal levels of 5-H.I.A.A. This result cannot be fully explained at present but it is probably connected with the fact that rectal carcinoids are atypical in their histology and behaviour when compared with their counterparts in the ileocæcal region. This case does not show cytoplasmic granules in all its tumour cells, and it is possible that this may be one reason why the level of urinary 5-H.I.A.A. remains within normal limits.

Further urine tests will have to be carried out on this patient at intervals to see whether the level of urinary 5-H.I.A.A. rises. Careful watch will also have to be made for any of the humoral manifestations of this disease. The whole question of the pathogenesis and behaviour of carcinoid tumours of the rectum is *sub judice* in view of the recent discovery that carcinoid is an endocrine tumour.

POSTSCRIPT.—This patient remains well to June 1956; there is no increased enlargement of his liver and the level of urinary 5-H.I.A.A. remains within normal limits.—**W. B. G.**

Pelvic Evisceration for Carcinoma of the Rectosigmoid.—E. G. MUIR, M.S.

I. B., aged 54, gave an eight months' history of lower abdominal pain, a discharge of pus *per vaginam* and cystitis. Cystoscopy showed a vesico-colic fistula and a barium enema a carcinoma of the rectosigmoid.

After a preliminary laparotomy and right transverse colostomy, pelvic evisceration was performed on 10.11.55. A carcinoma of the rectosigmoid had involved the bladder and the rectum. The bladder, cervical stump and upper part of the vagina were excised *en bloc*. Both ureters were implanted into the sigmoid colon and this was anastomosed to the rectal stump.

The patient made a good recovery, apart from some pelvic sepsis, and now awaits closure of her colostomy.

Tuberculous Disease of the Descending Colon with Previous Tuberculous Disease of Rectum Twenty Years Before.—E. G. MUIR, M.S.

H. S., aged 51, suffered from pulmonary tuberculosis in 1933 and later developed a fistula-in-ano which was treated at St. George's Hospital. Histology showed tuberculous disease. Further fistulæ formed and in January 1936 a left iliac colostomy was performed at King's College Hospital. Following this he remained in good health for nearly twenty years. In 1955 he commenced to pass blood and pus from the upper colostomy opening and was regarded for some time as ulcerative colitis.

Laparotomy showed a large mass in the descending colon and splenic flexure involving the spleen and tail of the pancreas. The mass and the colostomy opening were excised and the patient was left with a terminal transverse colostomy. The excised mass showed the histological picture of granuloma, presumably tuberculous, and the spleen showed amyloid disease.

Fibrosarcoma of Rectum.—W. B. N. BOMFORD, F.R.C.S. (for E. C. B. BUTLER, F.R.C.S.).

Mrs. E. L., aged 35.

Six months: Shooting pains in rectum and down left leg. No difficulty with defæcation.

On examination.—Pale, ill woman. *P.R.* Large, elastic tumour filling sacrum. *Sigmoidoscopy:* No ulceration of mucosa over large extra-rectal tumour.

28.9.55: Operation. Left internal iliac artery ligated. Synchronous combined abdomino-perineal excision. Large tumour in meso-rectum, attached to left wall of pelvis.

Made a good recovery.

Pathological report.—Pleomorphic, giant and spindle cell sarcoma from retrorectal region.

Follow-up clinic (2.1.56).—Progress satisfactory. No recurrence.

A Localized Stenosing Chronic Ulcerative Lesion of the Sigmoid Colon.—FRANK FORTY, F.R.C.S.

F. G., male, aged 47.

History.—Six weeks: blood and mucus *per rectum* and colicky pain. No significant physical signs.

Barium enema.—Smooth stricture 4 cm. long in sigmoid.

FIG. 1.

Operation.—Minimal thickening of bowel. Segmental resection—12 cm.

Specimen.—Sharply demarcated area of ulceration 4 cm. long, completely encircling the bowel. Wall thickened and œdematous (Fig. 1).

Section.—Mucosa destroyed. Ulcerated area covered by vascular granulations. Numerous lymphoid follicles in submucosa. All coats congested and œdematous.

Comment.—A rare lesion, which may be multiple or recurrent. Spontaneous resolution may occur. Laparotomy is usually required to exclude carcinoma. The treatment is segmental resection (Butler, 1953, *Proc. R. Soc. Med.*, **46**, 69).

Diverticulitis of the Cæcum Simulating Carcinoma.—H. JACK HARRIS, M.B., B.S.

A man aged 53 on examination by Mr. R. H. Gardiner in Aylesbury with reference to an old known duodenal ulcer, was found to have a mass on the right side of the abdomen. A diagnosis of possible carcinoma of the cæcum was made and at operation a matted mass of tissue was found involving the cæcum and ascending colon with attached portions of small intestine. The cæcum was stony hard and with difficulty a right hemicolectomy was performed, an ileo-transverse anastomosis completing the operation.

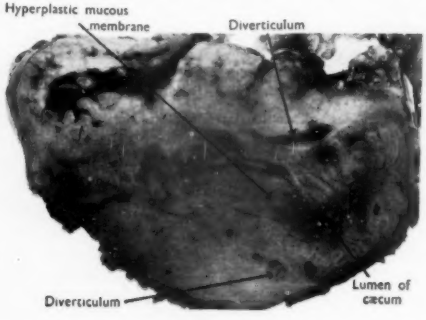

FIG. 1

Fig. 1 shows the appearance of the specimen when bisected in the laboratory, the differential diagnosis at this stage appearing to lie between, (1) carcinoma, (2) tuberculosis and Crohn's disease, (3) actinomycosis and (4) diverticulitis. A section was taken across the whole specimen and this showed the mucous membrane to be benign-looking and apparently hyperplastic. The thickening of the wall was composed of fibrous tissue while two sinus spaces could be seen lying outside the muscle coat and lined in part by large intestinal mucous membrane. This seemed to

confirm the diagnosis of diverticulitis of the multiple rather than the solitary form, there being at least two diverticula with no evidence of the presence of muscle in their walls. There were no further diverticula to be found in the ascending and transverse colon.

The interesting features of this case were, (1) the difficulties in making a diagnosis macroscopically both in the operating theatre and laboratory, and (2) the presence of multiple inflamed diverticula in the cæcum with no evidence of diverticula elsewhere in the large bowel.

Carcinoma of the Pelvic Colon Presenting with Vesico-colic Fistula.—P. T. SAVAGE, F.R.C.S. (introduced by W. W. DAVEY, F.R.C.S.).

Mr. P. T. Savage (Whittington Hospital) showed three surgically-resected specimens of carcinoma of the pelvic colon presenting with vesico-colic fistula.

In the first, from a man aged 58, a small fistula led to pain and frequency of micturition and pneumaturia. He was treated by combined partial cystectomy and pelvic colectomy. He is alive and well and free from recurrence after two and a half years.

The second specimen, from an old lady of 77, showed a huge vesico-colic fistula 1½ in. in diameter. She was treated by a three-stage resection of the pelvic colon, the involved fundus, posterior and left lateral wall of the bladder, uterus and left adnexa. The intramural portion of the left ureter was also resected and the left ureter implanted into the bladder. She died three months after closure of her colostomy, from massive recurrence in the vaginal vault, left internal iliac lymph nodes and liver.

The third specimen, from a man of 72, showed a bulky growth of the pelvic colon, invading the posterior wall and base of the bladder with a vesico-colic fistula ½ in. in diameter. He was treated by a combined total cystectomy and an extended Hartman's operation, with bilateral uretero-colic anastomosis. He died from a mild plastic peritonitis without evidence of a urinary leak, on the twelfth post-operative day.

The Radiological Diagnosis of Gastro-jejuno-colic Fistula.—A. LAWRENCE ABEL, M.S., F.R.C.S., and C. I. COOLING, F.R.C.S.

The case described was that of a male aged 66 who had developed a gastro-jejuno-colic fistula fifteen years after gastro-enterostomy for duodenal ulcer. The presenting symptom was intermittent diarrhœa for five months. Pictures were demonstrated of the barium enema X-ray, showing barium passing from the colon through the fistula into the upper gastro-intestinal tract. It was pointed out that the barium enema method is diagnostic, whereas a meal examination usually fails to reveal the fistula. The reasons for this were postulated. After surgical excision and gastrectomy, the stoma took thirteen days to function. The patient was home in three weeks and is now in good health.

FIG. 1.—Barium enema X-ray demonstrating free passage of barium through the gastro-jejuno-colic fistula, outlining stomach, duodenum and small intestine as well as whole colon.

Four Cases Illustrating Difficult Diagnosis and Complications in Colonic Disease.—J. M. ELSBY, M.B., B.S. (for HAROLD DODD, Ch.M., F.R.C.S.).

Case I.—J. S., male, aged 56.

History of two years' griping abdominal pain, and of six weeks' blood and slime *per rectum*. Second-degree hæmorrhoids found on examination.

Routine rectal examination normal, but with the left index finger in the right lateral position a large area of firmness was felt through the rectal wall. The barium enema would not pass the sigmoid colon. Sigmoidoscopy revealed an impassable stricture 1 in. above the recto-sigmoid junction.

Operation revealed a large mass, probably carcinoma, of pelvic colon adherent to terminal ileum. Pelvic colon and ileum resected and anastomosed (Fig. 1).

Histology.—Diverticulosis of colon, extending for 5 in.

Conclusion.—(1) The value of rectal examination with the left index finger. (2) The complexity and importance in the operative diagnosis between carcinoma and diverticulitis.

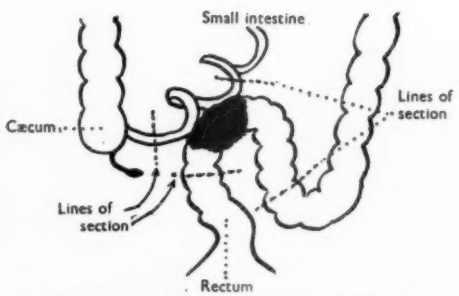

FIG. 1 (*Case I*).—Diverticulitis of pelvic colon adherent to terminal ileum. Ilium and pelvic colon resected and anastomosed end-to-end.

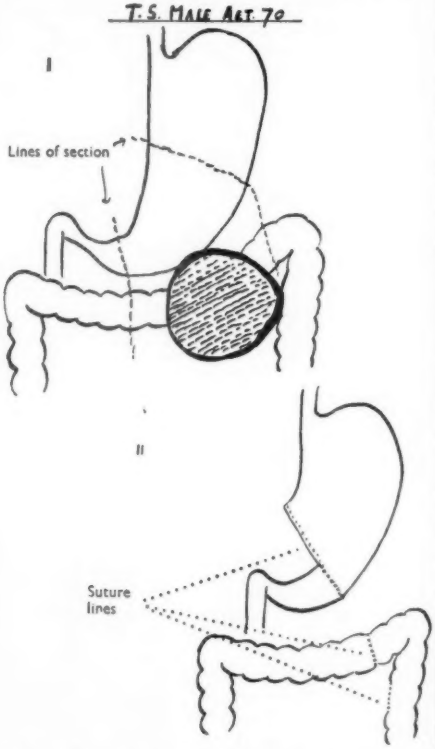

FIG. 2 (*Case III*).—I. Findings at operation. Globular tumour of transverse colon, 5 in. in diameter adherent to descending colon and greater curve of stomach. II. After resection and reconstruction.

Case II.—G. P., female, aged 69.

History of ten years' flatulence and excessive flatus. Bowels alternatively costive and loose. No blood or slime. Recent loss of weight. A mass was felt high on posterior wall. At operation two tumours were discovered, one in the pelvic, the other in the descending colon. Secondary carcinoma of the liver. Colon excised and anastomosed end-to-end.

Histology.—Lower lesion diverticulitis, upper lesion a differentiated adeno-carcinoma; eight lymph nodes show secondary carcinoma.

Conclusion.—A diverticular mass can mimic a carcinoma.

Case III.—T. S., male, aged 70.

History of six weeks' pain in the left loin.

Large, rounded, left subcostal mass which moved on respiration, was dull on percussion and it was possible to get above it. Occult blood in stools. Barium meal showed a filling defect in the body of the stomach due to a mass outside.

Operation revealed a tumour 6 in. in diameter in the transverse colon adherent to stomach and descending colon. Transverse colectomy performed, and Bilroth I gastrectomy, as the tumour could not be separated from stomach (Fig. 2).

Histology.—A globular tumour of the colon, 5 in. in diameter; a well-differentiated columnar cell carcinoma. Five lymph nodes involved.

Conclusion.—(1) An interesting diagnostic problem. (2) Operation complex, needing wide resection of stomach and colon. (3) Very large tumour is often slow to metastasize.

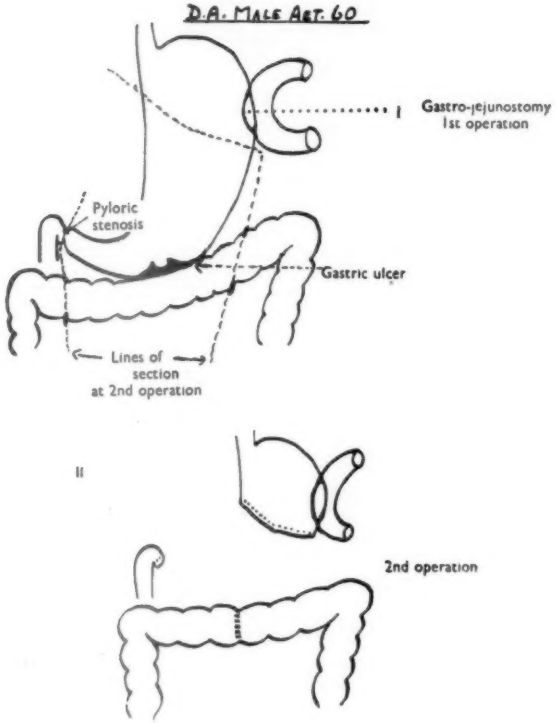

FIG. 3 (*Case IV*).—I. Findings at first operation. Pyloric stenosis, greater curve ulcer and gastro-enterostomy. II. After reconstruction at second procedure.

Case IV.—D. A., male, aged 60.

History of indigestion for thirty years. Five years ago hæmatemesis and melæna. Three months' anorexia, vomiting, flatulence, loss of weight. Pale, dehydrated, emaciated.

First operation, after partial correction of alkalosis, revealed pyloric stenosis and greater-curve ulcer adherent to transverse colon. Gastro-enterostomy (Fig. 3, I).

Second operation.—One month later ulcer larger and harder, clinically carcinoma. Partial gastrectomy and transverse colectomy (Fig. 3, II).

Histology.—Base of ulcer granulation tissue. No carcinoma.

Conclusion.—(1) Difficult differentiation between carcinoma and peptic ulcer. (2) Large ulcers on greater curve are usually carcinomatous. (3) Excision of transverse colon for adherent ulcer.

BOOKS RECENTLY PRESENTED AND PLACED IN THE SOCIETY'S LIBRARY

Ambrose (G. J.), and Newbold (G. F.). A handbook of medical hypnosis: an introduction for practitioners and students. pp. 255. London: Baillière. 21s. 0d. 1956.

Christopher (F.). Christopher's textbook of surgery. Edited by Davis (L.). 6th edit. pp. 1484. Philadelphia and London: Saunders. £5 8s. 6d. 1956.

Emerson (R. W.). Half-hours with great authors—Emerson. pp. 64. London, etc.: Raphael Tuck.

Francis (W. W.). W. W. Francis: tributes from his friends on the occasion of the 35th anniversary of the Osler Society of McGill University. pp. 123. Montreal: Osler Society. $5 (45s. 0d.). 1956.

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Tobias (E.). Periarteritis nodosa with special reference to the neurological complications. pp. 129. 's-Gravenhage: "Excelsior." 1956.

Todd (J. C.). Clinical diagnosis by laboratory methods. 12th edit., by James Campbell Todd, Arthur Hawley Sanford and Benjamin B. Wells. pp. 998. Philadelphia and London: Saunders. 42s. 0d. 1953.

Varrier-Jones (P. C.). Papers of a pioneer. Corrected by Peter Fraser. pp. 107. London, New York and Melbourne: Hutchinson. 1943.

BOOK REVIEWS

Office Procedures. By Paul Williamson, M.D. (Pp. ix + 412; illustrated. 87s. 6d.) Philadelphia and London: W. B. Saunders Company. 1955.

The title of this book may lead to some confusion in British minds, both professional and lay, unless it is realized that the term "office" has the same significance in the United States as "surgery" or "consulting room" in this country. An instance of this is the parson who picked up the book recently in the hope of finding some advice on the organization of an office. Instead he was confronted by the very graphic illustrations that form a major feature of the work and in fact are part of the text. His expression was ample evidence that they made their meaning clear to a non-medical mind.

This clarity is one of the virtues of the book and is compatible with the thoroughly practical approach which exists throughout it. The author has based the text on the notes which he has taken during his work as a practitioner to enable him to carry out a remarkably large number of minor practical procedures of varying complexity in all branches of medicine including the ear and the eye. A simple diagram for the recognition of which muscle is weak when diplopia is present is an instance of this. Explicit instructions for the treatment of various minor rectal disorders, and for the carrying out of simple gynæcological procedures are examples of the more important subjects which are described. Psychological testing, physiotherapy, the use of a small laboratory and some simple advice on radiology are included.

The critical attitude of the author in respect of results is very healthy, and he states that "most minor surgery we do gives poor results. By the standards of 1890 we do well: in terms of the knowledge we have to-day we do badly". He goes on to say that the cosmetic result could be improved by more attention to the basic principles in 75% of cases, and the morbidity in 50%. These principles he is at pains to detail.

Quite a number of the procedures which are described are usually undertaken in this country in the Out-Patient Department of a Hospital and not in the practitioner's own surgery. They are, nevertheless, so clearly explained that those practising at a distance from a Hospital may well find the book really useful, and certainly its possession will prove a source of support and comfort to the newly qualified. It is, however, a large book, and one that will usually remain in the surgery and not be part of a doctor's mobile equipment.

Cardiovascular Innervation. By G. A. G. Mitchell, O.B.E., T.D., M.B., Ch.M., D.Sc. (Pp. xii + 356; 217 illustrations, many in colour. 55s.) Edinburgh and London: E. & S. Livingstone Ltd. 1956.

It is rare to find such a detailed description of the nerve supply to the cardiovascular system so clearly portrayed as in this work. Not only are the peripheral ramifications of the sympathetic and parasympathetic nerves outlined, but their central connexions are also detailed both in the context and in outstanding illustrations.

The author has collected relevant material from the extensive literature dealing with this subject and has confirmed, augmented, or modified such views in the light of his own experience drawn occasionally from experiments in animals, but mostly based on his findings in man.

Clinicians who specialize in diseases affecting the separate systems of the body will find in this book a clue to many complex clinical syndromes based on the physiological variation or pathological alteration of the nervous supply to the vessels that feed the anatomical sphere which each specialty oversees. For this reason physicians should covet the possession of this valuable treatise on a subject hitherto never so comprehensively dealt with.

Arthroplasty. By St.J. D. Buxton, M.B., B.S., F.R.C.S. (Pp. viii + 126; 43 figs. 30s.) London: Pitman Medical Publishing Co. Ltd. 1955.

In this monograph, which will surely find a place in every orthopædic library, the author sets himself the double task of reviewing the progress made in arthroplasty during the last fifty years and of giving a lead for the future.

The historical section from Ollier and Murphy to the brothers Judet is full and comprehensive, and the indications, technique, and after-care of the operations which the author has found of the greatest help to his patients are well set out. His experiences with the Judet operation are described fully and the results of a series of one hundred such operations are subjected to a critical analysis. The conclusions reached will probably not find general

agreement, but the methods of assessment of results, which are set out in considerable detail, will undoubtedly encourage others to follow up their own results of similar operations with equal care.

The format is excellent and the numerous illustrations well reproduced. The full bibliography and index will prove of great value to all those who, like the author, feel that the restoration of controlled mobility to a stiff joint is one of the greatest benefits that operative surgery can bestow.

The Compend: a compendium of ethical proprietaries used in medicine and pharmacy. Compiled by W. Hetherington, F.P.S. (Pp. xxviii + 676. 32s. 6d.) Bristol: John Wright & Sons Ltd. 1955.

This compendium contains notes of some 2,000 ethical proprietary preparations of interest to medicine and pharmacy. Details are given of their manufacturers or distributors, their composition, therapeutic uses and dosage, and available packaging based on the manufacturers' literature. The assembling of this information must have entailed a great deal of careful work on all sides. The author has succeeded remarkably in getting it so nearly up to date, but the rate of development of new preparations is such that he has had to provide an appendix for those preparations developed or noted when the main body of the work must have been in the press. The arrangement of the preparations is alphabetical, but by the provision of a copy of the official list of approved names, and a guide to the individual monographs where active principles are listed and followed by their trade names, the task of finding a suitable preparation is made easier. There is also a useful list of international units and standard values mentioned in the individual monographs.

The author is to be congratulated on having gathered together all this information in such a handy and readable form. The reviewer for one is grateful to him for having performed a task so necessary, and hopes that the continuing increase in the therapeutic armamentarium will not discourage him from keeping this up to date in later editions when required.

The Relief of Symptoms. By Walter Modell, M.D., F.A.C.P. (Pp. x + 450. 56s.) Philadelphia and London: W. B. Saunders Company. 1955.

The author of this book feels that the rapidly increasing number of conditions for which a cure can be achieved, or at any rate attempted, has resulted in scant regard being paid to the relief of symptoms as such, to the considerable detriment of the patient. He has therefore set out to write about the relief of symptoms without laying emphasis on special treatments. This is a hard task because so often such relief is entirely dependent on the cure of the underlying condition, and this means that in places whole paragraphs of the book contain little more than general instructions to adjust electrolyte balance, or combat the processes underlying the fundamental disease.

There is, however, much to be found that is useful, and advice concerning the well-established methods of treating symptoms is given with common sense and, in places, with humour. The common-sense nature of the approach to a patient's problems is perhaps the most valuable part of the book as it enables the author to make clear the principle that the finding of practical forms of adjustment of the way of living results in the relief of symptoms at least as frequently as does the exhibition of drugs.

Twenty-four main symptoms have been selected for discussion, as these make up 95% of the complaints that bring patients to their doctors. Major problems, such as the relief of pain, anxiety, dyspnœa or œdema, and unconsciousness receive thorough discussion and detailed instructions for their treatment. The value of drugs is not, however, overstressed, but those of importance, such as opium, its derivatives and substitutes, receive full attention, the conclusions being much to the advantage of morphia.

The chapter on "Constipation" places the problem in its true perspective as it stresses the relative unimportance of this symptom, and the author's statement that "you are constipated if you think you are" makes his point of view clear.

Among the shorter chapters are ones on "Itching", "Hiccough", "Skeletal muscle spasm", "The masking of symptoms by cortisone" and on "Gas". This last comprises flatulence and flatus, and although nothing very much is recommended for its relief, it is interesting to read that American patients are less able to discuss this symptom with their doctors than appears to be the case in this country.

This is certainly not intended to be a textbook of therapeutics. There are no long lists of remedies of doubtful value, but the well-tried methods are discussed and described with clarity and discrimination. The author has brought to his task a pleasant manner of expression, an engaging habit of admitting when he does not know an answer, and a wide knowledge of medicine and of men.

Section of Anæsthetics

President—T. Cecil Gray, M.D., F.F.A.R.C.S., D.A.

[*January* 6, 1956]

Hypnosis for the Relief of Pain

By A. A. Mason, M.B., B.S.

No form of therapy has received at first such tremendous acclaim and then such universal condemnation as hypnosis. Every few dozen years there is a rise in popularity and great enthusiasm for its use, then follows a decline, often with widespread disapproval and hostility. The causes of these fluctuations are numerous and would form a fascinating lecture in themselves.

However, the most constant factor which has caused hypnosis to fall from favour so often, has been the way in which it is used rather than any intrinsic fault of the phenomenon itself. Patient and doctor alike search at one time or another for the "magic answer"; the powerful drug, vitamin or hormone to replace all others; the therapy, technique or antibiotic which is better than all others and which will cure resistant illness.

At first sight, hypnosis appears to fulfil this need perfectly. It is surrounded by mystery and awe, and appears to fulfil the prayers of misguided but fervent enthusiasts. These very enthusiasts, often well-meaning, do a great disservice to hypnosis, for they attempt to treat all manner of ills with it. Their claims are often exaggerated and highly coloured by their own unconscious desire for magic. Moreover, they often obtain results which appear on the surface to be dramatic and even highly important.

If one attacks a disease, even an organic disease, with great impact or "furor therapeutica", as Freud said, together with a deep belief in the infallibility of the technique, one may produce startling results. This is, of course, true for any kind of therapy one deeply believes in. When this therapy happens to be hypnosis, the very sound of which stimulates the patient's unconscious phantasies and desire for magic healing, the results may occasionally be devastating.

The literature abounds with the odd case of this or that which responded to the use of hypnosis, and never refers to the dozens of cases which did not respond. It is as though the hypnotist himself, believing in magic in the first place, cannot tolerate a percentage cure rate as with other forms of therapy, but must claim the infallibility which runs hand in hand with the omnipotent use of magic.

Again, hypnosis has been frequently employed by doctors (and laymen) who specialize in strange techniques and manœuvres unsupported by scientific investigation and probably depending largely on suggestion for their effect: odd rays, both ultra and infra; minute doses of drugs, or very large doses; ointments containing hormones, catalysts and active principles. They are the practitioners who cure everything by pulling on the neck or washing out the bowel; by a course of desensitization, or by "stimulating the phagocytes".

Now the diseases treated by these techniques are usually the asthmas, the migraines, the "rheumatics", the neurasthenics, the sensitive and the allergic. These diseases, while chronic and intractable, have one feature in common: they will respond favourably to any therapy initially. It is this initial improvement which so often appears in irresponsible literature, and less commonly the relapse which follows.

Hypnotism is also very efficacious in producing an initial improvement. However, when it is not followed by a management and understanding of the deeper and often the emotional factors in the disease process, it is as illogical to expect a cure from it as to expect an anæsthetic to cure appendicitis.

These factors have hitherto prevented hypnosis being investigated soberly and scientifically, and instead of a dispassionate judgment determined by careful and scientific research, it is first hailed as a panacea by its protagonists, and then discarded as useless by its antagonists. Neither valuation is correct; it is a phenomenon with real uses and equally real limitations, and as scientists and doctors we must so regard it. Until we can all use it with as little emotion and with as great facility as we use ether, we are not in a position to use it therapeutically, nor to assess its value.

Its Use in the Alleviation of Pain

Hypnosis has been used for the prevention and alleviation of pain from time immemorial. It was used before medicine was separated from the priests, and in the British Museum there is a bas-relief from a tomb in Thebes showing an Egyptian hypnotist and his patient some 3,000 years ago.

Æsculapius relieved pain by stroking with his hands, and even Hippocrates spoke of impressing health on the patient by passes.

Primitive tribes in the past, and still in the present, produce trance-like states in themselves by music or other rhythmical procedures. In these states, painful rituals, initiations and surgical operations are performed, apparently without pain.

In this country, two early exponents of hypnosis for operative analgesia were Squire-Ward who in 1842 amputated a leg under hypnosis; and James Elliotson, a senior physician at University College Hospital, who published a booklet in 1843 on numerous cases of surgical operations performed in the mesmeric state.

The third pioneer was James Esdaile, a graduate of Edinburgh, who was in charge of a native hospital at Hooghly, Bengal. He reported on 300 major operations and over 2,000 minor ones performed in the hypnotic state. He used natives he had taught to perform the hypnotism for him while he supervised. He received, however, little recognition in his time, probably because work originating from India was not considered seriously.

The Qualities and Properties of the Hypnotist

There is nothing supernatural or occult in hypnotism and, in fact, hundreds of doctors have learnt the technique successfully in the last few years.

The prestige of the hypnotist or that afforded to him by his patients is of considerable importance. The more senior the hypnotist, the greater is his success. Dignity, intellectual and social superiority and impressiveness are factors which increase his success rate. Confidence is certainly essential, the uncertain or inexperienced hypnotist being quickly detected by the perceptive unconscious of the patient. One should therefore discount the results of one's first two dozen cases and not be discouraged by frequent failures in them. To-day the modern hypnotist rarely follows the techniques employed by the older schools. These used dominating and aggressive methods and would often bawl in a loud voice "Go to sleep" to a cowering and intimidated victim, who would frequently oblige by falling into a profound trance. To-day such methods would produce only ridicule and laughter. The patient has to be soothed into a trance-like state slowly, gently, imperceptibly, often after having some explanation of the hypnotic state. It is not a technique for the impatient or hurried practitioner.

The susceptibility to hypnosis seems to have no relationship to the sex, race, profession or intelligence of the patients. Recently I was able to try the susceptibility of primitive Peruvian Indians to the use of hypnotism, and I found their response in most ways identical with highly cultured Englishmen. Broadly speaking, if the patient is accessible he can usually be hypnotized. Over-agitated, frightened and aggressive patients who cannot be made to co-operate are unfortunately inaccessible. Certainly the age of the patient is a factor of some importance, and the young are excellent subjects right down to the time when they first begin to understand language and simple instructions. 90% of all subjects attempted prove to be hypnotizable to some degree. It is of interest to note that the 10% of failures with one hypnotist may be hypnotized by another hypnotist, i.e. probably nearly 100% of people are hypnotizable by someone, which seems to imply that hypnosis is a normal, perhaps physiological, state which we all experience from time to time.

The techniques of hypnotizing patients can all be summarized in the following manner: They all depend on repeated rhythmical sensory stimulation carried on to the point of a sleep-like state. The stimulation may be auditory, a monotous voice or music; visual, changing lights, patterns or "passes"; or tactile, stroking. Common examples which all of us have experienced from time to time without being aware of their hypnoidal nature are as follows: When sitting in a train listening to the clickety clack of the wheels and watching the repeated telegraph poles going past the window our eyes often close involuntarily. When listening to a lecturer who speaks monotonously without variation in pitch or time an intolerable feeling of somnolence is frequently produced, as every student knows. The rhythmical rocking of the cradle by the softly crooning mother; the repeated stroking of the barber, of the masseuse, can all produce states of drowsiness and hypnoidal sleep. These common examples seem to show that the production of a hypnotic trance state is not pathological or mysterious, but a frequent occurrence, and one which we have all probably experienced or produced.

The method for the production of hypnosis which I have found most satisfactory is essentially that of progressive relaxation. I sit the patient in a chair, or lay him down on

a couch and repeat to him monotonously, rhythmically, frequently, suggestions of tiredness, relaxation, drowsiness, and the desire to sleep. I first instruct the patient to fix his eyes on a point above his head, merely for the purpose of keeping them from wandering and keeping his interest stimulated and to fatigue the retina with a constant image. In a little while, the patient's eyelids begin to get heavy, the eyes frequently water and then close. It is as simple as this. One could just as well start with the eyes closed, but their closure does give an indication of the production of the hypnotic state.

Other techniques include the use of music of monotonous nature to fatigue the auditory nerve; strokings and passes have all been employed; but on the whole, however, it pays to keep the technique as uncomplicated and simple to perform as possible. I do not advocate gazing directly into the patient's eyes. This may prove embarrassing at times, especially if one becomes hypnotized oneself first. It is essential above all to refrain from mystery and mumbo-jumbo. It is not necessary to have darkened rooms, mysterious lights and all the paraphernalia of stage technique. It is possible to use drugs to aid induction, the barbiturates having been widely used for this purpose. However, for analgesic purposes, to have to resort to barbiturates makes the whole procedure rather redundant. Scopolamine has also been used as an aid to induction. Hypnosis should be achieved in the average patient on the first try in approximately ten minutes. If I do not achieve hypnosis after two attempts I give up, for I feel both the unconscious resistance and the time necessary to overcome it make the procedure inexpedient.

Trance Depths

10% of patients are unhypnotizable by any single person. Obviously in these no analgesia is obtained. 35% of patients will go into a light trance, i.e. the eyes will close, they will respond to simple suggestions, but no analgesia is obtained. 35% of patients go into a medium trance in which there is analgesia, but it varies from slight (i.e. enough for dental fillings) to moderate (i.e. enough for orthopædic manipulations). 20% of patients go into a deep trance; considerable anæsthesia exists in this state.

Light trance hypnosis.—Although no analgesia exists in the light trance state, this level of hypnosis can find considerable use in the removal of the psychological overlay of pain. The patient who does not go to the dentist because of fear rather than anticipation of pain, the patient who is terrified of an anæsthetic either general or local, and in particular the patient who still feels "pain" despite adequate local or regional block. These are patients who are filled with fears and anxiety about any physical manipulation on themselves. Putting them in a light trance state and reassuring them in this state will often transform an unco-operative over-reacting terrified patient to the normal rather anxious but "determined to put up with it" average patient. Light trance hypnosis is an excellent premedication for general anæsthesia. It ensures a non-anxious and therefore smooth induction phase.

Medium trance hypnosis.—This can be obtained in 35% of patients, and is especially useful for repeated surgical procedures which may be painful but which do not warrant general anæsthesia. Needless to say, in the very ill or the young where general anæsthesia adds an additional burden to the patient's fight for survival, hypnosis may be valuable or even life-saving. Burn dressings, mastoid dressings, and painful ward dressings, all can cause great pain and discomfort, and can lead to a general lowering of morale and health. Repeated thoracic and abdominal explorations, repeated lumbar punctures, urethral instrumentation, orthopædic and physiotherapeutic manipulations, all these procedures can be painful and distressing. If this is so, they can be performed under hypnosis with the minimum discomfort. Multiple dental fillings, especially in children, frequently are easier to carry out under hypnosis than under locals. Even the minor procedures like sigmoidoscopy, gastric aspiration, antral puncture can cause anxiety and distress, and may be comfortably performed in the hypnotic state.

Deep trance hypnosis.—20% of patients can be made to enter the deep trance or somnambulistic level of hypnosis. Here analgesia is considerable. Amputations of limbs can be performed, removal of teeth, eye operations, ovarian tumours, Cæsarean section and appendicectomy have all been performed. (Photographs of a bilateral second stage mammaplasty performed by Mr. Matthew Banks under hypnosis were shown. The operation took 70 minutes and included excision of scars, fat and breast tissue and reshaping of breasts. The patient spoke, and drank fluids throughout the operation. There was no evidence of shock or other untoward reaction. The operation was witnessed by Dr. H. K. Ashworth and Dr. E. A. Bennet amongst others. (For a fuller report of this operation *see* Mason, 1955).

The advantages of deep trance hypnosis are as follows: there are no toxic effects of anæsthetic drugs, hypnosis may be of use in severely ill patients if chemical anæsthesia is contraindicated. Shock, always increased by the use of chemical anæsthesia, is thus minimized

by the use of hypnosis. The protective reflexes, apart from those which have been specifically removed by suggestion, are still present. Accidents are therefore less likely to occur. The patient would notice painful pressure or excessive heat in unanæsthetized areas and so ulnar pressure, brachial root traction, burning with hot fluids, would be brought to the notice of the surgeon by the patient. The cough reflex is unaltered, therefore blood or vomit cannot be inhaled (the latter accident being the commonest cause of anæsthetic deaths). Oral procedures are thus facilitated and rendered free from anxiety. The patient can drink fluids and so maintain fluid balance during a long operation. Pre-operative fear and anxiety can be relieved by suggestion and post-operative pain, discomfort and nausea can be prevented by post-hypnotic suggestion.

The disadvantages of hypnosis are its uncertainty of production, not more than 15% to 20% of cases being suitable for major surgical procedures, and the impossibility of forecasting suitable hypnotic subjects beforehand. Any emergency procedure is thus automatically ruled out.

Hypnosis and Burns

Crasilneck *et al.* (1955) report a successful series of treatment of severely burned patients with the aid of hypnosis. They give the following advantages: it is a method of anæsthesia which can be used for debridement, dressing changes and skin grafting. As it obviates the use of chemical anæsthesia, it takes some of the load off patients who are in pain, who are suffering from the toxæmia due to infected areas, and whose morale is at a low ebb due to the particularly demoralising effect of burned faces and hands. The repeated anæsthesias necessary in these patients are always a source of anxiety to anæsthetists and surgeons and, especially in children, may impede recovery[1].

Post-hypnotic suggestion may also be used to allay post-operative pain and allows the patient to become ambulant rapidly. The conversion of a supine dependent invalid to an erect person capable of motion, even though chronically ill, gives the patient hope that he will again become an independent person, and confidence that he will eventually recover from his injury.

Another severe problem in the burns patient is to maintain the food intake. Because of pain, depression, toxæmia and the nausea from analgesics and anæsthetics the appetite is poor and the patient will frequently not take in a normal calorific value. The open weeping and granulating surfaces of the burnt patient exude amino-acids and proteins and his calorie and protein needs are greater than normal. If he does not maintain a satisfactory intake level, the healing rate is retarded, epithelialization stops and skin grafts do not take.

In the hypnotic state, the appetite can be forced by the direct use of suggestion under hypnosis. Not only can this be done quantitatively but qualitatively and the patient can be given a specific appetite for high amino-acid-containing foods. Crasilneck reports elevating the calorie intake in his series of cases from 1,000 to 8,000 calories daily.

Hypnosis and Obstetrics

Hypnosis finds particular use in the relief of pain of parturition. Here one has many requirements to fulfil. One needs prolonged analgesia, sometimes for hours, sometimes for days. One needs a particularly non-toxic anæsthetic because of the length of the procedure. Most difficult of all, one needs an anæsthetic powerful enough to give relief of the severe pain of the second stage without depressing either uterine contractions or the infant's respiration. General and local analgesics are satisfactory in the hands of experts, but there are not sufficient of these to go round. Hypnotic analgesia is eminently safe, even in the hands of general practitioners or midwives, and often more potent than gas or pethidine which is all the latter have available. Many of the complications of childbirth are secondary to delay in one of its stages. The delay itself is frequently secondary to anxiety, fear or pain. All three of these symptoms can be relieved, wholly or partially, by the use of hypnosis, and delay and its sequelæ thus become correspondingly less frequent. The mother feels contractions, but no pain, and is conscious at the time of the birth of her child. This is said to have great psychological importance. Hypnosis is of special advantage in cases of toxæmia of pregnancy and cardiac failure where the mother is especially vulnerable to chemical anæsthesia.

The great G. B. DeLee (1939) wrote "The only anæsthetic without danger is hypnotism". Kroger and DeLee (1943) performed a series of 12 deliveries with 11 successes using hypnosis. Forceps and episiotomy were performed in all cases. They write: "The only method of relief of pain in childbirth that has complete safety for mother and baby without altering the normal medium of labour is hypnosis".

Perchard of the Mile End Hospital and Grasby of Pembury Hospital both report favourably on the use of hypnosis in obstetrics.

[1]F. T. Moore of East Grinstead reports on the use of hypnosis for burns dressing in a child aged 6.

I would finally like to point out the possible uses of hypnosis for the relief of pain in general medical practice. Although most specialist anæsthetists are concerned primarily with operative techniques, many are concerned with the relief of pain in patients whether it is surgically produced or a symptom of disease.

Hypnosis may be employed for the relief of pain whenever it is chronic, incurable and not dealt with adequately by the classical methods. Some examples of this type of use are as follows:

In inoperable cancer where pain relief is not adequately achieved with drugs. Butler, (1954) reports successful pain relief in 7 out of 12 inoperable cases of cancer. The patients have to be fairly good hypnotic subjects to get relief from severe pain and if they are deep trance patients they can be taught "auto-hypnosis" and so bring about relief of pain themselves.

The pain of conditions like trigeminal neuralgia, post-herpetic neuralgia, causalgia and phantom limb pain may be helped by the use of hypnosis in two ways. Firstly, it is possible that the actual number and severity of the attacks can be diminished by direct hypnotic suggestion. Secondly, the patient may be taught auto-hypnosis so that he has a method always to hand of producing pain relief himself when it is needed.

The physiological mechanism which is responsible for the production of analgesia in hypnosis is not very clear. It is thought by some to be allied to distraction anæsthesia, by some to be a blocking of pain impulses at the synaptic junctions. However, one thing may be said positively, and that is that the patient being operated on under hypnosis shows no elevation of pulse, change in blood pressure or respiration, i.e. there is no evidence of central appreciation of the pain, or of shock. So although the mechanism by which pain is diverted from cortical appreciation is unclear, it seems plain enough that it happens.

There still remains an enormous field open for research of all kinds into the uses of hypnosis. No subject needs it more and none has had so much unscientific nonsense talked about it. I feel it is time the anæsthetists in particular gave it a better position in their books and teaching. It is a method of producing analgesia and, as such, they as specialists in this branch of medicine should be acquainted with its technique. Lots of time and energy are spent in learning the properties of chloroform and it is hardly ever used. Hypnosis, which could be of considerable use, is not taught at all.

Finally, I would like to say that I believe hypnosis is going to find respectability and its place in medicine at last. It is being evaluated by groups of doctors in all branches of the profession, and that dangerous anachronism the "specialist hypnotist" will vanish, to be replaced by specialists in all branches of medicine who use hypnosis as just another weapon in their medical, psychiatric or anæsthetic armoury.

[The lecture was followed by a demonstration of induction of hypnosis, the production of anæsthesia and auto-hypnosis.]

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Dr. H. K. Ashworth said he was much obliged by the invitation from Dr. Mason to be present as an independent observer at the operation just described for the excision of bilateral extensive mammary scars under hypnosis, and also for the facilities afforded to examine the patient. Hypnosis, in this case, had been a spectacular and unqualified success, and no other form of anæsthesia had been necessary.

On examination, shortly before operation, the patient was walking about her bedroom, enjoying a cigarette. She had no pre-medication, nor, by her demeanour, was there any reason to suppose that she herself had taken any kind of soporific. Her pulse rate was then 94. On the operation table before induction of hypnosis her pulse rate was 128, B.P. 124/80, respirations 24. During the operation one of the striking features of the proceedings was the entirely normal conversation which the patient carried on from time to time with Dr. Mason on extraneous matters.

At the end of the operation the patient was quite comfortable, pulse 100, B.P. 140/80, respirations 24. Ten minutes later B.P. had fallen to 130/80.

The patient was re-visited the same evening. She was slightly excited and her pulse rate was 120, but this seemed a natural reaction to an experiment which, by then, she realized had been a complete success. She made no complaint of pain at the site of operation. She also had no recollection of having conversed with Dr. Mason during the operation and even showed some signs of embarrassment when one of the subjects of conversation was mentioned. It seemed quite clear that amnesia during the period of hypnosis had been complete.

During the operation postural hypotension was used, but no hypotensive or local anæsthetic drugs were injected.

Hypnosis alone provided perfect anæsthesia for the operation performed.

Dr. A. Spencer Paterson pointed out that the technique of hypnosis was related to the physiology of the autonomic nervous system, which differed from the central nervous system in being under the control of the imagination rather than of the will. For instance, it was not possible to get a patient to make his pulse go fast or slow by exercising his will power, but under hypnosis one could influence the rate of the pulse by putting a picture into the patient's mind of it slowing, or becoming more rapid. Hypnosis could influence the autonomic nervous system in other ways as well, such as in raising a blister. It was not surprising, therefore, that it had been able to influence skin diseases, while in the anæsthetic sphere it had been possible not only to inhibit pain, but to induce the patient to maintain a calm attitude. An interesting line of investigation would be to record changes in the autonomic nervous system effected by hypnosis, changes which could not be effected by exercise of the will.

Dr. A. A. Mason, in reply to various speakers, said that in an obstetric case it was, of course, useless to attempt to treat a new case once it had gone into labour. The case should be seen on several occasions before labour commenced and conditioned to respond rapidly to hypnotic suggestion.

The line of investigation that Dr. Spencer Paterson suggested was in fact being carried out by various people and interesting results were forthcoming. It seemed that certain people only could respond with their autonomic nervous system to direct suggestion. It was possible that these autonomic responders were the patients who develop psychosomatic disorders as distinct from psychoneurotic disorders when faced with similar stress situations.

[*March* 2, 1956]

Posture During Anæsthesia [*Abstract*]

By Ronald Jarman, D.S.C., D.A., F.F.A. R.C.S.

This paper pointed out the importance of attending to the comfort of the unconscious patient, and noted that the literature contained few references to the subject.

Slides and a colour film were shown to demonstrate, in detail, the various positions in which a patient might be placed on the operating table, and the hazards peculiar to each position were discussed.

Methods of avoiding pressure sores, bruises and nerve palsies were considered, and reference was made to diathermy burns and to the ease with which unsupported muscles, ligaments and joints may be strained—especially when the muscle relaxant drugs were used.

It was stressed that too much care could not be exercised by the anæsthetist in in helping to avert these complications.

The Management of Children Undergoing Tonsillectomy [*Abstract*]

By F. R. Gusterson, B.Sc., M.B., B.S., F.F.A. R.C.S.

A child entering hospital may suffer severe and lasting emotional upset unless properly handled. The film shown at the Meeting emphasized that doctors, nurses and parents all had their part to play in preventing such emotional trauma.

The points emphasized were:

(1) *The mother* should prepare the child for entering hospital by explaining the hospital routine. She should bring the child to hospital and stay with it on the day of admission. A favourite toy should accompany the child.

(2) *Nurses* must realize that they will act as "mother" to the child. As few nurses as possible should handle the child in hospital. Masks should not be worn over the face until the child is asleep.

(3) *Premedication.*—The barbiturate group of drugs had been used for many years but had certain grave disadvantages, the chief of which were their uncertainty of action and the danger of respiratory depression. Methylpentynol caused no respiratory depression, was easy to administer and had a large margin of safety. Fear and apprehension were abolished.

A technique had been evolved in which hyoscine was dissolved in elixir of methylpentynol, so avoiding the pre-operative injection of atropine.

The operation of tonsillectomy was chosen as an example but the remarks would apply equally to any operation, other than those on the stomach or upper alimentary tract.

Observations on Steroid Anæsthesia

A Preliminary Report

By R. P. HARBORD, M.D., F.F.A R.C.S., and W. N. WILD, F.F.A. R.C.S.

Hans Selye (1942) reported on a study in animals of the effects of large doses of steroid hormones, substances which were insoluble in water. Little effect was noted from solutions in oil given subcutaneously. To obtain a more rapid effect he tried the intraperitoneal route and produced a deep anæsthesia sufficient to allow for long abdominal operations. Complete recovery followed. Many steroids were studied without revealing any correlation between chemical structure and anæsthetic activity.

Laubach *et al.* (1955) investigated a steroid known as 21-hydroxypregnane-3, 20 dione sodium succinate (hydroxydione), which can be given intravenously as it is readily soluble in water or mildly alkaline buffer solutions. A comparison in different animal species showed a potency equal to, or slightly less than, thiobarbiturate, the therapeutic index being higher for hydroxydione. No endocrine activity was found in animals after large doses.

Murphy *et al.* (1955) reported on the effects of the Viadril brand of hydroxydione in man. They believed it had "analgesic" properties because they used less pethidine and relaxant drugs with hydroxydione than with a barbiturate, but the figures for the drugs used with the barbiturate are omitted; this makes their evidence unacceptable. They state that 1·0 to 1·5 grams of hydroxydione appears to have its maximum effect for about two hours with nitrous oxide and oxygen but do not reveal how they came to this conclusion. The presence of nitrous oxide confuses the issue, as we shall presently show. They took elaborate precautions and used a 2·5% solution, injecting it into the tubing at some distance from the arm with the stopcock fully open. The transfusion needle was inserted into the median antecubital vein and the injection into the (5% dextrose in water) drip made at the rate of 1·5 grams in five minutes. They found thrombophlebitis in 3 patients who had widespread vascular disease, but we are left to guess at how many of their 125 patients had clear evidence that the veins used only for the drug did not become thrombosed, for thrombosis is common after transfusion.

Taylor and Shearer (1956) reported on their work in animals and in 130 human subjects. In animals they show how irritating the drug could be to veins and also how an animal, deeply under the effects of hydroxydione, would react to external stimuli. In man they stated that 0·5 gram did not usually result in unconsciousness; 1·0 gram produced a deep coma, but in spite of this the patient would make some movement on stimulation which could be prevented by nitrous oxide and oxygen. They argued, therefore, that the drug was more hypnotic than analgesic. They noted the fall in blood pressure and rise in pulse-rate, and made the generalization, without declaring the evidence, that after 1·0 gram, one would expect an average adult to be awake and rational but still drowsy at the end of 1½ hours. They used in the main a 0·5% solution, giving it at the rate of 1·5 grams in from 2 to 5 minutes, into the tubing of a 5% dextrose saline. They found venous thrombosis in 15 out of 70 patients and this they regard as the essential drawback to the use of the drug.

Howland *et al.* (1956) state that since deep sleep produced by hydroxydione—with evidence of electroencephalographic level four—is insufficient to abolish reflex reaction to painful stimuli, it is therefore a non-analgesic hypnotic. Increasing the dose can lead to severe hypotension. They think 1% solution may be irritating to veins; 2½% is very irritating.

Finally they argue that many of the widely used anæsthetics can be given so that the patient has a rapid awakening without having to have a prolonged induction or the risk of hypotension or phlebitis, as with hydroxydione.

We have deliberately restricted the number of patients in order to make it clear how we have come to our conclusions. We preferred to do this rather than to produce a large series with little or no data, giving instead our private interpretation of the results.

Four examples show how patients reacted to hydroxydione when the operative risk appeared to be considerable.

Case I (J. H.) was a man aged 78 years who had an abdominoperineal resection of rectum immediately followed by a prostatectomy. During the 3 hours' operating time and for 1¼ hours after disconnecting the anæsthetic apparatus, the systolic blood pressure remained above 100 mm.Hg. He had artificial respiration, nitrous oxide and oxygen inhalation; 0·7 gram hydroxydione, 200 mg. Flaxedil, 50 mg. Scoline (for intubation), but no pethidine. He made a good recovery.

Case II (L. U.) was in poor general condition from the ravages of ulcerative colitis. She was pyrexial (101° F.), and had developed mental symptoms shown by persistent "rambling". Her blood pressure was normal (135 mm.Hg), but a tachycardia of 128/minute was recorded before operation. A total colectomy lasted more than 2 hours and she received 0·5 gram hydroxydione, 50 mg. Scoline (for intubation), 320 mg. Flaxedil but no pethidine. She had no hypotension (80 mm.Hg).

Case III (C. L.) had developed cerebral thrombosis the day after an anterior resection of rectum conducted with chlorpromazine, pethidine and Phenergan. He developed a chest complication and tracheotomy was performed. When he burst his abdomen several days later, and when he appeared to all about him to be moribund and in a state of semi-consciousness, hydroxydione was given in dose of 0·4 gram. He had no pethidine or Scoline but had 40 mg. Flaxedil, nitrous oxide and oxygen and artificial respiration. He did not have hypotension (80 mm.Hg) during or immediately after operation. He died two days later still in a coma.

Case IV (E. R.), aged 85 years, died while on the operating table immediately following an anterior resection of rectum. The heart stopped 3½ hours from the initial and only hydroxydione injection which amounted to 0·5 gram. Almost immediately before death she had Prostigmin to counter the effects of 160 mg. Flaxedil (given in divided doses). She had 50 mg. Scoline for intubation but had no pethidine. Hypotension first developed 2½ hours after the initial hydroxydione. Cyanosis of the face occurred early on when artificial respiration was applied by pulmoflator with positive and negative phases but disappeared on reverting to the manual method. At autopsy she was found to have a large pulmonary embolus.

It can be argued that similar results could be obtained by other methods but our data shows that hydroxydione, if carefully managed, offers promise when the risk to life is high.

In our small series of 25 patients, 13 were males. The general range of age in all 25 was from 26 to 85 years, 18 being between 40 and 60 years. 16 of the operations were major abdominal procedures. The range in operating time was from 20 minutes to 3¼ hours. Premedication was mainly (17 instances) with pethidine and hyoscine given intramuscularly. The Viadril brand of hydroxydione was given in divided doses, the totals ranging from 0·4 to 2·1 grams; 12 having between 0·6 and 1·0 gram. Pethidine was used in doses ranging from 50 to 200 mg., but 15 had none. Scoline, usually in doses of 50 mg. was used for intubation. Hydroxydione is said to have relaxant properties but Flaxedil was given in doses ranging from 40 to 360 mg.; 8 patients had more than 200 mg. It was given for various reasons: (1) to produce muscular relaxation, (2) to reduce the effects of hiccup, and (3) to maintain a state of apnœa. Artificial respiration was applied in all the abdominal operations. Nitrous oxide and oxygen alone was given by inhalation in all but 3 patients.

Induction was started in most instances with 0·5 gram hydroxydione and was uneventful; there being no struggling. The shortest period from the initial injection to surgery was 16 minutes. Two patients stated that the colour of objects had changed. One said that the ceiling was revolving, but most could not remember the period immediately after the injection. Some became more nervous after the initial injection because they expected to go off to sleep immediately. We found that intubation after 0·5 gram hydroxydione was not a practical procedure, and preferred to use relaxants to get ideal conditions. One patient complained of pain during the initial injection. Generally, the superficial veins appeared to remain about the same width as before the initial injection.

With 3 exceptions all the patients became unconscious after 0·5 gram hydroxydione as judged by their failure to respond to our shouting "open your eyes". Fig. 1 shows the examples where 0·5 gram resulted in unconsciousness. The majority became unconscious within 5 to 10 minutes from the initial injection.

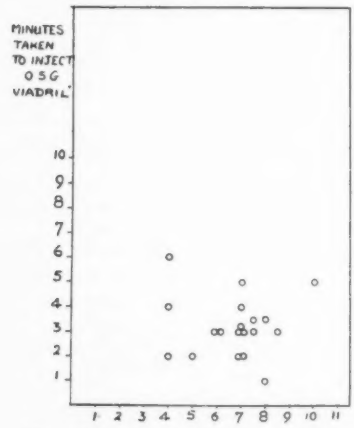


FIG. 1.—Rate of onset of unconsciousness.

It appears that with doses of 0·5 gram there is no relation between the onset of unconsciousness and the speed of the injection. We are at a loss to understand the statement by Taylor and Shearer (1956) that 0 5 gram did not usually produce unconsciousness.

In 9 patients hiccup-like spasmodic respiratory movements were seen. In 3 patients they persisted for periods over 20 minutes. The longest period was 40 minutes. In 2 the spasms began before the peritoneal cavity was opened. The movements appeared to be greater in extent than those occurring with other anæsthetic combinations. Treatment was with more hydroxydione, pethidine or Flaxedil or by a combination of these, and in one instance by squeezing the reservoir bag vigorously and rapidly. The movements could be reduced but frequently recurred.

Changes in blood pressure and pulse-rate during surgical operations due to hydroxydione are not easy to analyse but in the series there were 5 with hypotension of the order of 80 mm.Hg. No vasopressor drugs were used and the hypotension lasted for more than 20 minutes in only 2 patients. One of these was the patient with a pulmonary embolus and the other will be described later. Only one patient had a transient hypotension in the immediate post-operative period. We thought that the general condition of patients at this stage was better than after other anæsthetics but were at a loss to know how to prove it.

The initial effects of hydroxydione on blood pressure before any movement of position and before surgery is shown in Fig. 2 which reveals a general tendency for it to fall, though not to hypotensive levels. Fig. 3 shows that this change was generally accompanied by a rise in pulse-rate which did not exceed 110/minute.

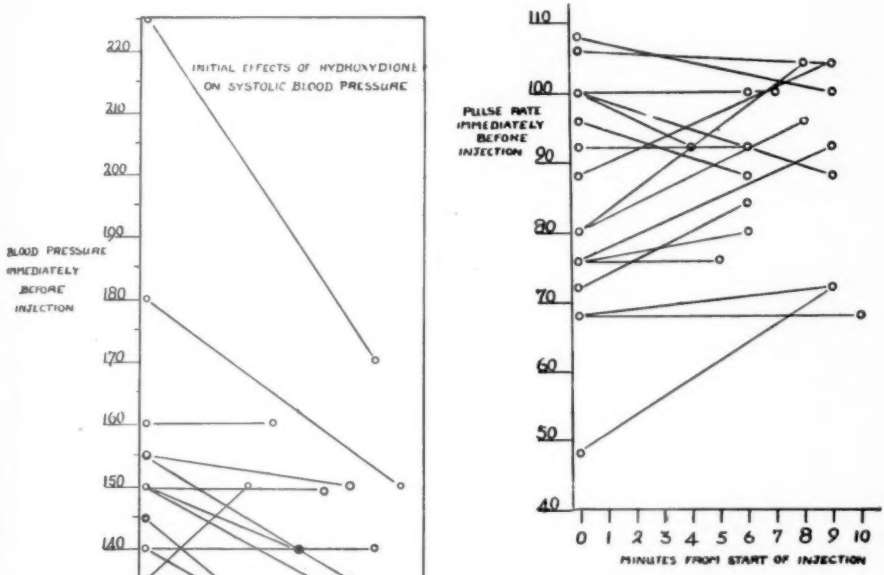
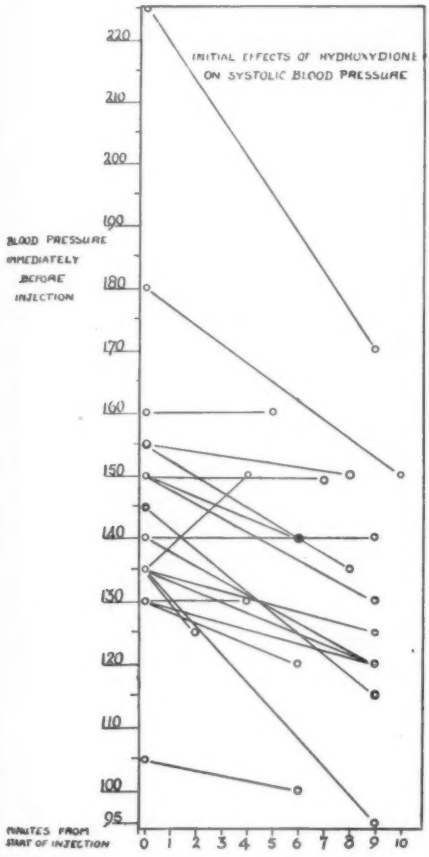


FIG. 3.—Initial effects of hydroxydione on pulse-rate.



← FIG. 2.—Initial effects of hydroxydione on systolic blood pressure.

Fig. 4 shows the blood-pressure change in a patient breathing air lying on his back without any disturbance by the surgeon or others. The fall in systolic pressure is marked and is accompanied by a fall in pulse-rate.

There are other points of interest in this record: (1) During the period between the initial injections of hydroxydione at 0 and 72 minutes when the Scoline was given, the effects must have been due to hydroxydione. He became unconscious at 7½ minutes. He "jumped" when the transfusion needle was inserted at 8 minutes, and a withdrawal reflex occurred on stroking the foot at 13 minutes. He then continued to have occasional spasmodic movements which did not seem to bear any relation to outside stimuli, although he would move on stimulation. The readiness with which this patient moved on stimulation does not mean that this drug has no analgesic properties. Had more of the drug been given he might have remained still. (2) Note the disturbance of blood pressure and pulse-rate when the herniorrhaphy begins. (3) Note the short operation and short inhalation which in this instance was confined to oxygen. (4) Note the long period from disconnecting the anæsthetic apparatus to the return of consciousness. It would be safe to argue that the period of unconsciousness was due to hydroxydione, for he had not had pethidine. (Pethidine is generally regarded as an analgesic drug but we have observed a conscious patient to

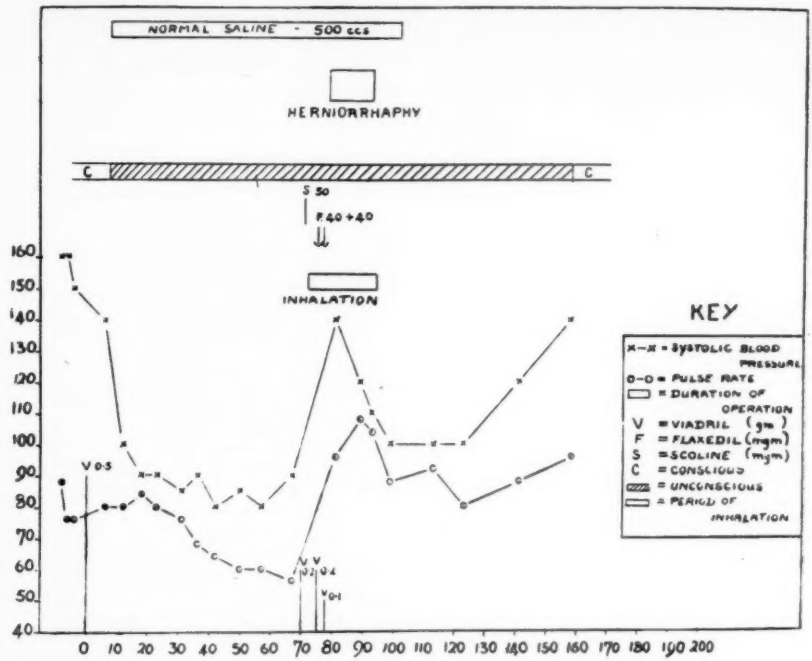


FIG. 4.—Chart of patient having Viadril with oxygen inhalation.

become unconscious after 50 mg. pethidine, given intravenously immediately following operation.) This patient, aged 60 years, had 1·0 gram hydroxydione and was unconscious for a period lasting 2 hours 38 minutes. Compare this response with that of another patient (*see* Fig. 5), aged 42 years, also having a hernial repair operation with 1·0 gram hydroxydione—this time with nitrous oxide and oxygen. The total period of unconsciousness was 2 hours 40 minutes. It looks as though 1·0 gram hydroxydione lasts about 2½ to 2¾ hours.

Fig. 6 shows the period in minutes between the end of inhalation and the return of consciousness after 20 operations. It reveals a rough correlation between the dose and the period of unconsciousness. Consciousness was judged by the ability of the patient to obey commands. In more than half the examples the period of unconsciousness was terminated within 10 minutes from the end of the inhalation. All this means is that in about half of the patients the inhalation of nitrous oxide and oxygen was terminated at periods when the effects of hydroxydione in producing unconsciousness had worn off.

It is of interest to note that 3 of those having 0·5 gram had periods of unconsciousness fairly near together. They were 1 hour 38 minutes, 1 hour 55 minutes, and 1 hour 22 minutes.

From this analysis showing the wide range of the responses it will be seen that statements about the effect of hydroxydione in the "average patient" are apt to be far from the mark.

We used three techniques: (1) 0·5 gram hydroxydione was dissolved by 50 c.c. saline (normal) giving a 1% solution which we injected into a vein. (2) The same percentage of hydroxydione was given via the drip saline, and (3) 2½% hydroxydione was also given via the drip saline. Where a drip was used it was our practice to wash through with a large amount of saline after the injection and also we injected 10 c.c. of saline after the drug when using the vein direct. With method (1) the veins used for the drug were palpated and found to be hard in 8 out of 15 examples. 2 out of 3 with method (3) had no sign of hardened veins, and in method (2) we found the evidence unsatisfactory. Thrombosis is a disadvantage but we intend to go on using more and more dilute solutions until a safe concentration is found.

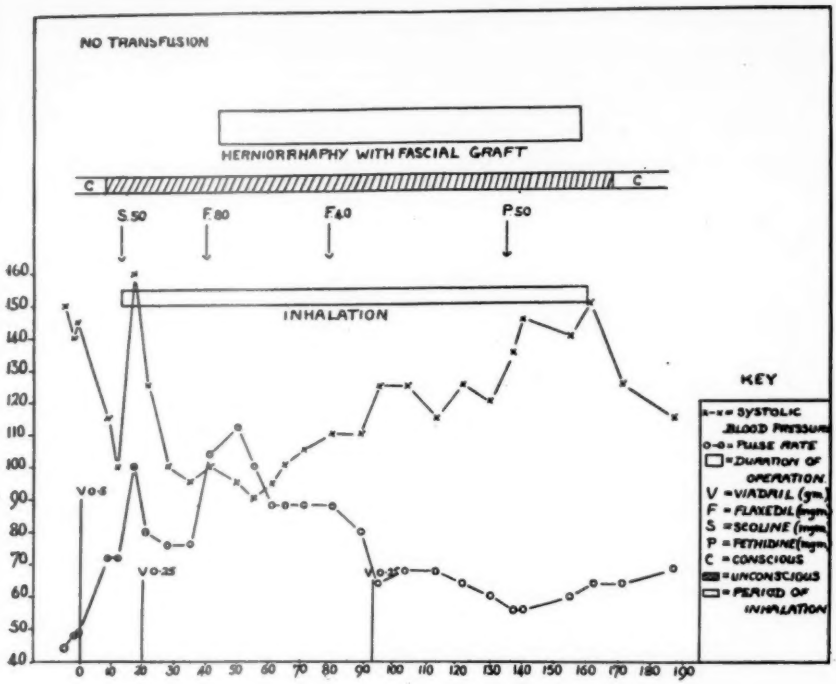


FIG. 5.—Chart of patient having Viadril with nitrous oxide oxygen inhalation.

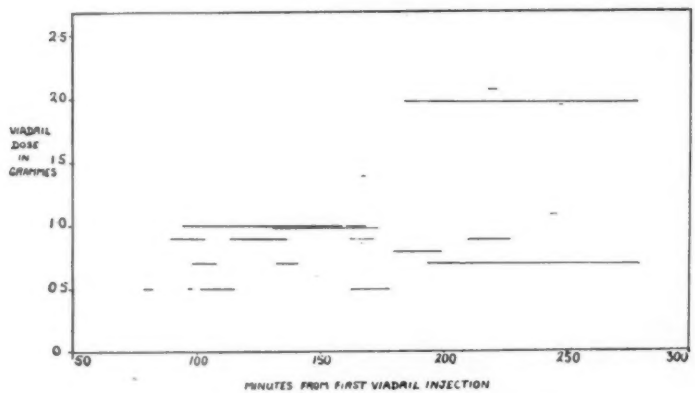


FIG. 6.—Lines show end of inhalation to return of consciousness.

We are at present exploring the possibility of using other combinations. In the first we induce with thiopentone and continue with hydroxydione. In another we premedicate with chlorpromazine and continue with hydroxydione.

It is our impression that hydroxydione depresses respiration. We have found that patients readily developed apnœa when we began to compress the reservoir bag intermittently. We did not find difficulty in getting the patient to breathe spontaneously at the end of operation and used Prostigmin on twelve occasions.

SUMMARY

Points from the literature on steroid anæsthesia have been discussed. An investigation of 25 patients showed how operations can be conducted under steroid anæsthesia in those who are seriously ill from various causes. Unconsciousness was produced in most instances by 0·5 gram hydroxydione. The blood pressure fell and the pulse-rate rose slightly during the induction. About one-third of the patients developed hiccup during upper abdominal operations. Notes were made of the duration of unconsciousness. Thrombosis of the veins used for hydroxydione injections was a disadvantage. Our observations would tend to show that this drug offers promise and further investigation is proceeding.

ACKNOWLEDGMENT

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Adrenal Response to Stress Before Operation, During Anæsthesia and During Surgery

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IT has become well established that one response of the body to stress is stimulation of the adrenal glands to secrete various corticosteroids (Selye, 1952). The functions of these compounds have been only partially elucidated, and the mechanism for the performance of these functions is even less well understood. However, one of the primary values of the adrenocortical hormones is mobilization of energy for cellular work. Sayers and Sayers (1949) suggested that adrenal hormones have a supporting rather than an initiating role in bodily processes. It is known that cortisone may alleviate the pains of rheumatoid arthritis; it is known that the 11-desoxycorticosteroids exert a special influence on electrolyte metabolism. Adrenocortical insufficiency is characterized by a negative sodium balance. Renal tubular absorption of sodium and water is inadequate in the absence of adrenocortical hormones. This situation is seen in Addison's disease. Under these circumstances the volume of extracellular fluids is not maintained, and the concentration of extracellular sodium is decreased. Osmotic forces then move water to intracellular sites. With the hypovolæmia which follows, blood viscosity and plasma protein concentrations increase. Cardiac output and renal and peripheral blood flows diminish, and a shock-like crisis may ensue. Desoxycorticosterone and aldosterone are especially potent in offsetting this sodium and water imbalance. Renal absorption of sodium requires energy for transference of sodium from tubular urine to tubular cell. The increased renal excretion of sodium that accompanies adrenocortical insufficiency can be explained therefore on the basis of submaximal mobilization of energy.

Cortisone and compound F have little effect on electrolyte or water metabolism, but they are highly active in carbohydrate metabolism. A deficiency of these oxygen-containing hormones results in rapid depletion of glycogen reserves. This action is probably integrated with protein metabolism in that adrenocortical insufficiency diminishes gluconeogenesis. Administration of compound F enhances the transformation of protein to glucose. The (protein) catabolic effect of adrenal hormones can be prevented by administration of glucose or can be accentuated by insulin. It seems then that the hormone causes these changes when necessary; i.e. the action is a supporting, not initiating, role.

Since surgical procedures may involve considerable stress, it seemed desirable to attempt to obtain some measure of evaluation of the various factors involved during the operative experience. Accordingly, measurements of free plasma dihydroxycorticosteroids were chosen as an indication of adrenal response to trauma. Compound F is the steroid present in the blood of man in the greatest quantity (Romanoff *et al.*, 1953), and is measured in this determination.

Method

Blood samples were taken from 10 patients two days before surgery and from 15 patients the day before surgery. They were taken from these 25 patients and from 22 additional patients immediately before anæsthesia, after one hour of anæsthesia, and again after one hour of surgery. In some cases samples were drawn at the end of surgery and again on recovery. Analysis was done by the Nelson-Samuels method (Nelson and Samuels, 1952). this method can be relied upon to measure accurately levels of dihydroxycorticosteroids within a range of less than 3 micrograms per 100 c.c. of plasma. It involves extraction of the blood with chloroform, partitioning extraneous material, chromatography, and final development of a colour with phenylhydrazine and sulphuric acid. Changes in values of blood corticosteroids appear within fifteen minutes after a stressing reaction, so an hour of anæsthesia was deemed adequate to measure the effect of the anæsthetic agent. There were 3 groups of patients on the basis of the anæsthetic agents used; 10 received nitrous oxide and thiopental (thiopentone), 10 received cyclopropane only, and 27 received ether. Of the patients receiving ether, anæsthesia was induced in 10 with nitrous oxide, in 9 with thiopental and cyclopropane, and in 8 with cyclopropane alone.

Results

Table I shows that the average corticosteroid concentration of the samples taken from 10 patients two days before surgery was 9 gamma/100 c.c. plasma, and immediately before surgery that group had a level of 12·7 gamma/100 c.c. The average plasma steroid values of the 15 patients taken one day before surgery was 12·7 gamma, and immediately before surgery their average was 14·6 gamma. The changes recorded were not statistically significant.

Table I.—Plasma Dihydroxycorticosteroids Before Anæsthesia

No. of patients	2 days pre-operatively γ/100 c.c.	1 day pre-operatively γ/100 c.c.	Immediately before anæsthesia γ/100 c.c.	Critical ratio
10	9*		12·7	1·727
15		12·7*	14·6	0·579

Differences not statistically different.

*Uncorrected for diurnal variation.

Table II.—Plasma Dihydroxycorticosteroids During Anæsthesia and Surgery

No. of patients	Agent	Pre-anæsthesia γ/100 c.c.	One hour of anæsthesia γ/100 c.c.	One hour of surgery γ/100 c.c.	End of surgery γ/100 c.c.	Recovery
10	Nitrous oxide-thiopental	12·7	13·1A	19·8B	30 x	39 x
10	Cyclopropane	12·4	12·5	16·3		
27	Ether	13·7	18·5C	29·2D	32 y	39 z

x 6 patients. A–B *
y 15 patients. C–D ***
z 13 patients.

*The difference between the figure marked A and that marked B is statistically significant.

***The statistical difference between the figure marked C and the figure marked D is very highly significant.

Table II indicates that the plasma concentration of corticosteroids during one hour of nitrous oxide-thiopental or cyclopropane anæsthesia was essentially unchanged. The average value during ether anæsthesia was somewhat elevated but the increase was not statistically significant. 11 of the 27 patients, however, responded to ether anæsthesia with increased values of 10 gamma or higher. The other 16 patients showed little or no such response. The average blood corticoid values rose during the hour of surgical manipulation, and this increase could be correlated with the degree of trauma. The cyclopropane series was done with patients having short surgical procedures so data beyond 1 hour of surgery are unavailable. Little or no increase of blood corticoid values was apparent during these minor procedures. The increase observed during the hour of surgery in the nitrous oxide-thiopental cases is made up of (A) very little change in those patients having minor procedures and (B) a fairly large increment observed in the patients whose surgery was more extensive. These data are supported by the higher values observed in the smaller number of patients from whom data are available beyond the one hour of surgery in the more lengthy procedures. Most of the patients receiving ether had fairly extensive surgery, and the continued elevation of steroid levels consequent to the trauma was consistently found.

Continuing rise of steroid levels at the time of recovery (samples usually taken at 4.30 p.m.) should also be noted. This was observed consistently regardless of the anæsthetic agent.

Discussion

There is evidence that a diurnal variation occurs in plasma corticosteroid levels (Tyler *et al.*, 1954; Engel *et al.*, 1955; Doe *et al.*, 1954), the early morning values being about 5 gamma/100 c.c. higher than levels measured later in the day. Even without taking this into account, no statistically significant variation was found in comparing samples taken two days and one day before surgery with those taken immediately before anæsthesia. When correction for the 5 gamma/100 c.c variation on control samples taken later in the day was made, the average values the day before surgery are even slightly higher (no significant difference) than the controls taken the morning of surgery. These results are definitely contrary to those observed by Franksson and Gemzell (1955) who reported significant differences in 33 patients on the two days leading up to surgery. They obtained average values of 5·5, 8·5 and 14·5 gamma/100 c.c. on the three consecutive days. Franksson and Gemzell's samples were taken at the same time each day. Their control values were lower than ours. So far as can be ascertained, their samples were not partitioned during the analyses. The control values determined on samples drawn immediately before starting anæsthesia in our series had an overall average of 13·2 gamma/100 c.c. which agrees well with those found by Samuels' group (Sandberg *et al.*, 1954) who found normal values of 13 gamma/100 c.c. on morning samples.

The unchanged values during nitreous oxide-thiopental anæsthesia are consistent with the observations of Moore's group (Vandam, 1955) who found essentially no rise with these agents. They did, however, report increases when cyclopropane was the agent used. No such change was observed in this series. No opinion regarding this difference can be advanced until the details of their work become available.

The results obtained during ether anæsthesia indicate that ether may sometimes be a factor causing adrenocortical stimulation and sometimes not. 11 of the 27 patients showed definite rise of corticosteroid levels, but 16 gave no such increase. One can only speculate at this stage of knowledge concerning reasons for this difference in behaviour.

By the end of surgery the reaction to surgical trauma had overshadowed differences due to anæsthesia. Plasma corticosteroids during the recovery period rose to even higher values than those found at the end of surgery. It is impossible to say whether the stimulus to production of these compounds ended with closure of the incision, or whether the bodily processes involved in healing may have provided a stimulus. The half-life of infused hydrocortisone in plasma has been measured at 1·0 to 1·5 hours (Peterson *et al.*, 1955). On this basis, if recovery values were determined four hours after the cessation of surgery, it would seem that elevated levels must have been due to continuing production of hormones by the adrenal glands.

Summary

(1) Dihydroxycorticosteroids of blood have been determined before and during anæsthesia, and during and after surgery as a measure of bodily response to stress.

(2) Values obtained two days and one day prior to surgery were not significantly different from those found immediately before surgery.

(3) Blood steroid levels were not elevated during an hour of cyclopropane anæsthesia, nor during an hour of nitrous oxide-thiopental anæsthesia.

(4) 11 of 27 patients receiving ether for an hour showed an elevation in blood corticosteroid level.

(5) Blood steroid levels rose during surgery in direct relationship to the intensity of the surgical procedure. They continued to rise for several hours, the highest level being reached some time after surgery had been completed.

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